Final Report

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Prepared for

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Acronyms and Abbreviations

AFS	Absorption Fraction
ALM	Adult Lead Model
AT_c	Averaging Time for Carcinogenic Effects
AT_{nc}	Averaging Time for Noncarcinogenic Effects
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	Bioaccumulation Factor
BCF	Bioconcentration Factor
BKSF	Biokinetic Slope Factor
BW	Body Weight
C	Fish Tissue Concentration Corresponding to Target Risk
CCC	Criterion Continuous Concentration
CDC	Center for Disease Control
Cu	Copper
CWA	Clean Water Act
DI_x	Dietary intake for chemical x
DO	Dissolved Oxygen
ED	Exposure Duration
EF	Exposure Frequency
ER-L	Effects Range-Low
FC_x	Concentration of chemical x in fish
FIR	Food Ingestion Rate
GSDi	Geometric standard deviation PbB
I	Intake
IEUBK	Integrated Exposure Uptake Biokinetic model
IR	Fish Ingestion Rate
IRIS	Integrated Risk Information System Database
KSD	Mass fraction of soil in dust
LOAEL	Lowest Observed Adverse Effect Levels
MCL	Maximum Contaminant Limit
MGBC	Machine Gun Boat Coarse
MRL	Minimal Risk Level
NA	Not available or not applicable.
NEPA	National Environmental Policy Act
NOAEL	No Observed Adverse Effect Levels
NPDES	National Pollution Discharge Elimination System

OME	Ontario Ministry of Environment and Energy
Pb	Lead
PbB	Blood lead
PbB0	Baseline PbB
PHA	Preliminary Health Report
PRG	Preliminary Remediation Goal
RfD	Reference Dose
SAV	Secondary Acute Value
Sb	Antimony
SCV	Secondary Chronic Value
TEC	Threshold Effect Concentrations
THI	Target Hazard Index
TR	Target Risk Level
USCG	United States Coast Guard
USEPA	U.S. Environmental Protection Agency
VPIT	Vessel Posing Imminent Threat
WC_x	Concentration of chemical x in water
WIR	Water Ingestion Rate
WS	Weighting factor; fraction of IRS+D ingested as outdoor soil
Zn	Zinc

Executive Summary

This Preliminary Health Report (PHA) evaluates the human and ecological risk assessments of proposed U.S. Coast Guard (USCG) live gunnery training. USCG cutters and boats are in the process of being outfitted with small arms (machine gun and/or shoulder fired rifle). Live gunnery training will be conducted to develop and evaluate the ability of boat crews to correctly and precisely apply small arms fire against a Vessel Posing Imminent Threat. The metals that constitute the bullet have the potential to affect human health and the environment. The PHA evaluated risks to human health and the environment from the ammunition discharged into water bodies during training activities.

The overall approach of the PHA followed standard risk evaluation procedures and used "realistic worst case" assumptions. For example, the risk was calculated assuming all rounds fired would be the largest rounds available and the maximum number of rounds required in any situation would be used in all situations. Three types of environments where the potential for risk is greatest were evaluated: freshwater lake systems, with the Great Lakes as the representative; estuarine systems with Chesapeake Bay as the representative; and a riverine system. The analysis was generic in that it assumed typical biological, chemical and physical conditions for the representative areas but did not include site-specific factors such as existing sediment concentrations of metals. The PHA identified two areas where anticipated risks could be elevated: areas near potable water intakes and depths shallower than 20 feet. These areas will be avoided during training exercises and thus the risk to human health and the environment was not quantified for these two sensitive areas.

A conceptual model was developed that describes the exposure scenarios that may lead to human health and environmental effects. Discharged ammunition will be deposited in the sediment within training areas, and the metals that constitute the bullet might dissolve into sediment pore water or adsorb to the surrounding sediment. Plants and animals that exist at the bottom of the food chain might ingest and be directly exposed to the metals in sediment. In turn, fish might ingest these plants and animals and accumulate the metals in their body tissue, which is then consumed by recreational fishers, birds, or mammals.

Based on USCG training procedures, characteristics of the ammunition, properties of the sediment and surface water in each the environmental types, and using standard geochemical and bioaccumulation models, concentrations of the metals that comprise the bullets (i.e., antimony, copper, lead, and zinc) were estimated in sediment, surface water, and fish tissue. Exposure parameters (e.g., fish ingestion rates) were then compiled and used to calculate exposure concentrations for recreational fishers, birds, and mammals. The predicted exposure concentrations were then compared to effect levels for human health and environment. The effect levels were obtained from the scientific literature and regulatory sources. Risk estimates were determined using the screening risk quotient method. Screening risk quotients are calculated by dividing the exposure concentration by the effect level. Screening risk quotients that exceed one indicate the potential for risk because the concentration exceeds the effect level. However, effect levels and exposure

concentrations are derived using intentionally conservative assumptions. Risk quotients greater than or equal to one do not necessarily indicate that risks are present or impacts are occurring. Rather, it identifies risks requiring further evaluation. Risk quotients that are less than one indicate that risks are very unlikely, enabling a conclusion of no significant elevated risk to be reached with high confidence. The maximum screening quotients calculated for any media are shown in the table below.

Table E.1
Maximum Human Health and Environmental Screening Quotients for USCG Training Exercises[1]
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Bullet Constituent	Plants, Invertebrates, Fish	Birds and Mammals	Recreational Fishers
Antimony	0.58	NA	0.04
Copper	0.75	0.05	0.33
Lead	0.96	0.14	0.39
Zinc	<0.01	<0.01	<0.01

[1] The threshold effect level is 1.0 (i.e., values greater than 1.0 indicate that further investigation is needed)

Predictions of sediment, surface water, and fish tissue concentrations of antimony, copper, lead, and zinc did not exceed human health and ecological effect levels in the training area. As previously stated, the PHA used "realistic worst-case" assumptions, which were intended to be conservatively protective of human health and ecological receptors as well as provide for reasonable limitations on training activities. Based on the results of this evaluation, proposed training will result in no elevated risks for the Great Lakes/freshwater, estuarine/Chesapeake Bay, and riverine systems scenarios, and further investigation is not recommended.

Subsequent to preparation of the PHA, risks in marine sediments from live gunnery training were qualitatively evaluated. Risks in the marine water column were not considered significant because analyses for the other settings (i.e. lakes, estuaries, and rivers) conclusively showed no elevated risk in water or sediments, with predicted concentrations in sediments closer to criteria. Thus if there was any indication of elevated risk in marine settings, it would appear in the sediments before the water column. Risks in the marine sediment were considered similar to other environments and with no indication of elevated risk.

1.0 Introduction

This Preliminary Health Assessment (PHA) Report evaluates the human health and ecological risk assessments of proposed United States Coast Guard (USCG) live gunnery training over open waters. USCG cutters and boats are in the process of being outfitted with 50- and/or 60-caliber machine guns. To evaluate the ability of boat crews to correctly and precisely apply small arms fire (machine gun and/or shoulder fired rifle) against a Vessel Posing Imminent Threat (VPIT), proper training and judgment is necessary to reduce the risk of collateral damage on confined waterways in and around industrial or urban areas.

The discharge of metals associated with the bullets from the weapons has the potential to affect human health and the environment. In order to assess potential effects the USCG has contracted the preparation of this PHA which addresses both human health and ecological risk. This PHA is generic in that it does not address individual specific sites, but it is comprehensive in that it evaluates potential effects in three types of environments where potential for risk is greatest: Great Lakes/freshwater systems, estuarine systems (Chesapeake Bay used as a prototype) and a riverine/lotic system.

The overall objective of this PHA was to evaluate potential risks to human and ecological receptors associated with expended small caliber munitions from USCG live gunnery training. This evaluation intended to answer one of three general questions for each risk scenario evaluated:

- Are significant elevated risks predicted using "realistic worst case" assumptions?
- Are no elevated risks predicted even with "realistic worst case" assumptions?
- Is there an unacceptale level of uncertain, such that unanticipated occurances result in elevated risks?

Based on the results of this evaluation, proposed training will result in no elevated risks for the Great Lakes/freshwater, estuarine/Chesapeake Bay, and lotic systems scenarios using "realistic worst case" assumptions, and further investigation is not recommended.

1.1 Objectives and Overall Approach

The overall objective of this PHA is to evaluate potential risks to human and ecological receptors associated with expended small caliber munitions from USCG live gunnery training. The evaluation of risks relies on modeled concentrations in sediment, surface water, and tissue as determined from estimates of annual expenditure of ammunition, physical and chemical characteristics of surface water and sediment, and standard models, as described in the following sections. The characterization of risks involves identifying the potential exposures of receptors expected near the training exercises and evaluating the potential effects associated with such exposures.

The overall approach of the risk evaluation follows standard procedures, as described below, and uses "realistic worst case" assumptions. These assumptions are described in the

in Section 1.3. The result of this evaluation will answer one of three general questions for each risk scenario evaluated:

- Are significant elevated risks predicted using "realistic worst case" assumptions?
- Are no elevated risks predicted even with "realistic worst case" assumptions?
- Is there an unacceptale level of uncertainty, such that unanticipated occurances result in elevated risks?

Based on the outcome of this evaluation, recommendations will be made regarding the need for additional investigations, including the initiation of a Baseline Risk Assessment (BRA), if necessary.

1.2 General Description of Risk Assessment Process

The methods and approaches followed in this report were adapted from applicable U.S. Environmental Protection Agency (USEPA) guidance (e.g., USEPA 1997a, 1998). The components of risk assessment include development of a conceptual model that identifies and evaluates potential source areas, transport pathways, fate and transport mechanisms, exposure media, exposure routes, and potential exposed receptors. The remaining components include an exposure assessment, effects assessment, and risk characterization. The principal activity associated with the exposure assessment is the estimation of chemical concentrations in applicable media to which the receptors might be exposed. The exposure assessment involves estimating exposures to potential receptors for the exposure scenarios identified. The principal activity associated with the effects assessment is the development of chemical exposure levels that represent conservative thresholds for adverse effects. The risk characterization portion uses the information generated during the exposure and effects estimates to calculate potential risks for the exposure scenarios evaluated. Also included is an evaluation of the uncertainties associated with the models, assumptions, and methods used in the PHA, and their potential effects on the conclusions of the assessment.

1.3 Human Health and Environmental Protective Assumptions

As stated in Section 1.1, the overall approach uses "realistic worst-case" assumptions. This approach is intended to be conservatively protective of human health and ecological receptors as well as provide reasonable limitations on training activities. The assumptions used in this PHA can be divided into two types: approach and operational. Details of operational assumptions (e.g., minimum and maximum body weights for receptors) used in this PHA are described in the applicable sections. Approach-related assumptions are those that describe the scope of the PHA, and are listed below.

- Training activities will not be conducted in the vicinity of the most senstive areas, such as potable water intakes, national wildlife refuges, and national parks. The location of these areas should be considered prior to initiation of training exercises, and avoided, if possible.
- Existing or background contamination from previous activities is not considered, and therefore, current risks can not be evaluated. The risk evaluation will only

consider future risks due to training in the hypothetical test firing areas. Training exercises that take place in areas with existing levels of contamination with the potential for cumulative impacts with expended ammunition are not considered in this PHA. As long as the existing sediment concentrations are not already near or over screening levels used in this PHA, the addition of metals from USCG training operations will not result in elevated risk. Even if existing concentrations are elevated above screening concentrations, USCG operations should not result in a measurable elevation of risk.

- Training exercises will not be performed at depths shallower than 20 feet (ft.). Risks associated with training activities at depths shallower that 20 ft. are considered unreasonably high due to the high probability of upper-level trophic receptor direct contact with expended ammunition.
- Location specific factors such as critical habitat, special-status species, or potable water intakes were not identified or considered due to the hypothetical nature of the study area. These factors should be considered prior to initiation of training exercises and avoided if possible.
- Five years of training activity are evaluated. Risks associated with training activities that extend beyond this five-year period are not evaluated in this PHA.

The uncertainties associated with these assumptions are discussed further in Section 8.0.

2.0 Conceptual Model

Figure 3-1 illustrates the conceptual model for the PHA. Important components of the conceptual model are the identification of exposure scenarios, the source, transport pathways, exposure media, potential exposure routes, and potential receptor groups.

Exposure refers to the potential contact of an individual with a chemical. The exposure assessment identifies pathways and routes by which an individual may be exposed to the constituent metals and estimates the magnitude, frequency, and duration of potential exposure. An exposure pathway can be described as a mechanism that moves a chemical from its source to an exposed population or individual, referred to as a receptor. An exposure pathway must be complete or exposure cannot occur.

As shown is Figure 3-1, discharged ammunition will be deposited in the training area. The metals that constitute the bullet might dissolve out of the ammunition into pore water and either adsorb to the surrounding sediment or become suspended in the overlying surface water column. Lower-trophic level receptors (benthic invertebrates, water-column invertebrates, and fish) might ingest and be directly exposed to metals in surface water and sediment. Metals in sediment, surface water, plants, and invertebrates might be consumed by and accumulated in the fish tissue, and thus be transported to upper-trophic level receptors (recreational fishers, birds, and mammals) via food webs. The analysis considered possible consumption of infaunal organisms, which have a greater exposure than epibenthic organisms. Thus, if upper trophic level organisms or humans did consume epibenthic organisms, their exposure would be less than the infaunal consumption assumed in the model.

2.1 Great Lakes/Freshwater System and Chesapeake Bay/Estuarine System

The conceptual model shown in Figure 3-1 is applicable to the Great Lakes/freshwater system and Chesapeake Bay/estuarine system. Specific receptors, as well as surface water and tissue concentrations, differ between the two systems, and are described in more detail in Section 4.0.

2.2 Lotic System

The conceptual model for the lotic/riverine system is the same as that for the Great Lakes/freshwater system. Specific receptors, as well as sediment, surface water, and tissue concentrations, are considered similar, such that risk conclusions and assumptions for the Great Lakes/freshwater system also apply to slow-moving riverine systems. For this reason, further evaluation of the lotic system was not performed. Training activities conducted in areas of high current and high deposition immediately downstream have the potential to produce areas of deposition unaccounted for in the current analysis. However, targets are not anchored, and high current areas would result in potentially rapid movement of the target, hindering the training exercises. For these reasons, areas of high current areas and high rates of deposition immediately downstream should be avoided for training exercises.

3.0 Source

As shown in Figure 3-1, discharged ammunition can deposit on the sediment and metals can dissolve into pore water and adsorb to sediments. For this evaluation, the discharged ammunition was conservatively modeled as sediment (i.e., the weight and chemical composition of the discharged bullets was assumed to be sediment with an equal weight and chemical composition). This assumption is an overestimate of the actual contribution of metals to the aquatic system because metals will dissolve out of the bullets and adsorb to sediments at much lower levels. This assumption is discussed further in Section 8.0.

3.1 Method for Calculation of Bullet Density

Bullet density was determined from characteristics of the ammunition, sediment, and training procedures.

3.1.1 Ammunition Characteristics

USCG training exercises include both 7.62 mm and 0.50 cal ammunition. Since 0.50 cal ammunition will be used in training exercises conducted by Port Security Units only (approximately 20,000 rounds discharged annually), there will not be over water training creating a potential risk to the aquatic environment. Thus, risks from discharge of only 7.62 mm were investigated in this PHA. The highest intensity of training will occur at Great Lake Stations, so the rate of ammunition use at these stations was used as a realistic worse case. There are 43 stations in the Great Lakes District and each station could qualify as many as 10 gunners. Since each gunner trains by discharging 1000 rounds annually, the deposition could be as high as 43,000 rounds of 7.62 mm per station annually by the USCG Ninth District (USCG, personal communication, Table 3-1).

For the purposes of this investigation, it was assumed that only bullets, and not cartridges, were discharged into the water body. There are two types of 7.62 mm bullets. The bullet with the greatest mass (149 grains or 9.7 grams; Table 3-1) was selected for modeling purposes as a conservative (i.e. protective of human health and the environment) assumption.

3.1.2 Training Procedures

The USCG training exercises are described in the Machine Gun Boat Coarse (MGBC) Exercise Framework (USCG, draft report, upublished). The MGBC Exercise Framework was created to allow different small boats in the Coast Guard inventory an opportunity to perform a standardized form of machine gun training. Boats are maneuvered along a coarse that simulates target vessel course/speed changes (Diagram 1 in the MGBC), allowing gunners to fire at a buoyant and stationary target when they have an un-obscured firing bearing. Maximum firing distance to the target is 400 yards, and the minimum firing distance is 50 yards.

3.1.3 Depostional Area

The size of the depositional area was calculated from a description of the training activities in the MGBC Exercise Framework and several conservative assumptions. For modeling purposes, the quantity of discharged ammunition was divided evenly among 12 areas. Each of the 12 areas consisted of a 30 degree angle originating from a firing point along the course and a length of twice the distance between the gunner and the target (Figure 3-2). The sum of each these 12 areas made up the Total depositional area. Within the Total depositional area, 30% of the bullets were expected to be deposited within a Target Area. To account for bullet skipping along the water surface, the Target Area was defined as 100-meter radius around a central point. A surface drift of 0.25 knots was conservatively used to determine the total deposition area (USCG, personal communication) and resulted in a 200-yard change in the starting course and target location. Since a drift away from the previous firing stations resulted in a larger depositional area than if a drift occurred towards the previous firing area (Point Alpha to Point Bravo), the Total and Target areas were adjusted to account for 1/3 of the training exercises occurring with a drift away from the maneuvers (i.e. the larger area) and 2/3 occurring in the smaller area.

3.1.4 Sediment Conditions

Values for sediment density and depositional depth were necessary for calculating a sediment mass over the Total and Target areas that would be mixed with the discharged ammunition. A default value of 2.40 g/cm^3 was used for sediment density in the Great Lakes (Kemp et al. 1977; Table 3-1). Sediment mixing depth for deposited bullets was assumed to be 5 cm (Table 3-1). Additional sediment characteristics necessary for determining pore water concentrations are described in Section 5.2.1.

3.2 Predicted Bullet Densities in Total and Target Areas After 5 Years

Estimated bullet densities in the Total and Target areas after 5 years are shown in Table 3-1. To account for cumulative deposition over the five year training duration, annual expenditures were added together to calculate the Total area bullet density. It was assumed that the Target area would be in the exact location for each training event every year. However, the calculation was made assuming that the target area would shift, from year to year, but would be within the previous year's Total depositional area. Sediment concentrations are the same for both freshwater and estuarine systems.

A list of the key conservative assumptions used in the source characterization, their effect on the risk estimate, and more realistic assumptions are presented in Table 3-2.

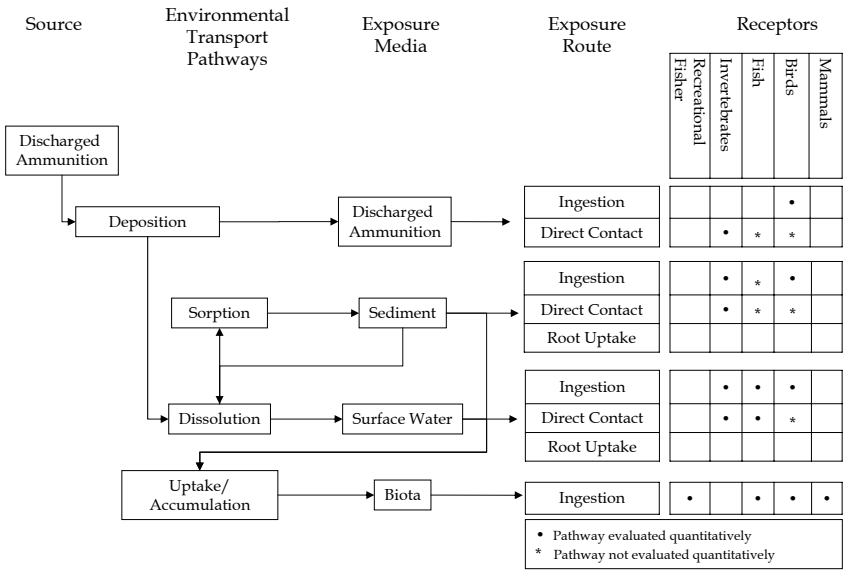


Figure 3-1. Preliminary Conceptual Site Model

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

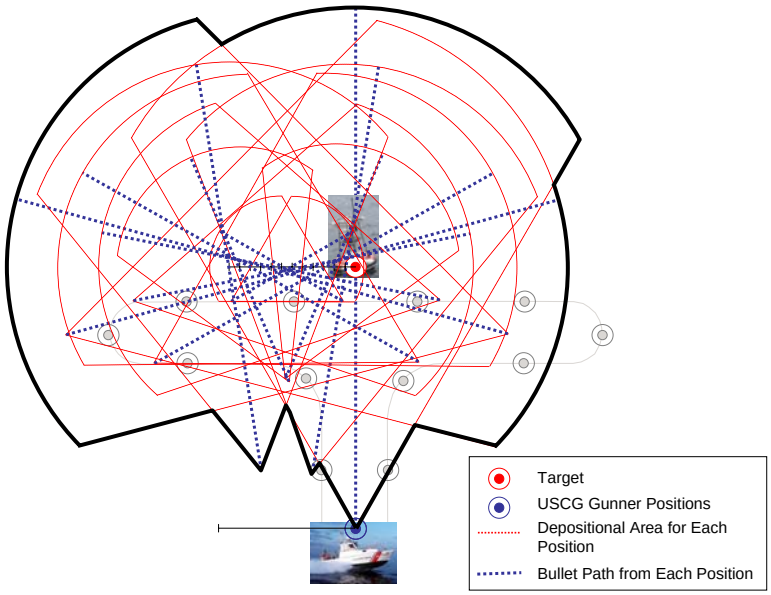


Figure 3-2. Total Depositional Area Based on Drift in One Direction (Note: the area represented is 427,505 m^2. The area used for evaluation was 389,086 m^2 and was based on a weighted average of right and left drift)

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Table 3-1
Source Characteristics and Bullet Density
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Source Parameter	Value
Ammunition Characteristics	
Total No. 7.62 mm Rounds Fired Annually - All Stations in USCG Ninth District	430,000
No. of Stations in USCG Ninth District	43
Max. Weight of Individual 7.62 mm Round (mg)	9,674
Max. Antimony Per Round (%) [1]	6.8%
Max. Copper Per 7.62 mm Round (%) [1]	31%
Max. Lead Per 7.62 mm Round (%) [1]	76%
Max. Zinc Per 7.62 mm Round (%) [1]	3.5%
Sediment Characteristics	
Sediment Depth (cm)	5.0
Sediment Density (g/cm^3)	2.4
Target Area Size and Bullet Density	
Size of Depositional Area (m^2)	27,943
5-year Bullet Density (mg/kg)	98.16
Total Area Size and Bullet Density	
Size of Depositional Area (m^2)	389,086
5-year Bullet Density (mg/kg)	10.360

[1] The total percentage is not 100% because values are the maximum allowable in manufacture of two 7.62 mm bullet types

Table 3-2
Key Realistic Worst-Case Protective Assumptions Incorporated into the Source Characterization
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Protective Assumption	Realistic Scenario	Protective Assumption Effect on Risk Estimation
All qualifiers fire rounds of maximum weight (i.e., 7.62 mm vs. 5.56 mm) and at maximum discharge rate (i.e, 500 rounds semi-annually vs. 100 or 150 rounds for shoulder-fired weapons)	1/3 of qualifiers on machine gun fire 500 7.62 mm rounds semi-annually; 1/3 on rifle fire 100 7.62 mm rounds semi-annually; and 1/3 on carbine fire 150 5.56 mm rounds semi-annually	Concentrations over estimated up to 2 times
Target located at same exact location all 20 events each year	Little overlap in target locations within a year (estimated at 5-10%)	Concentrations over estimated proportional to location variation
All bullets confined to top 2 inches of sediment	Bullets could be found in up to 6 inches of sediment	Concentrations over estimated proportional to bullet settling > 2 inches
Assumes no sediment mixing, deposition or migration	Sediment mixing, deposition, and migration variable, and at times significant	Concentrations over estimated proportional to sediment dynamics
Depositional area the same in each year	Small overlap of depositional areas (25-30%)	Concentrations over estimated proportional to yearly variation in training area
Only 1 skip and no underwater travel assumed for target area	Bullets skip several times and continue to travel underwater past the target area	Concentrations over estimated proportional to bullet travel
Minimal time and drift velocity assumed for training exercises	Time and drift velocity at minimal for half the exercises and greater for remainder of exercises [1]	Concentrations over estimated proportional to time and drift velocity

[1] Training activities conducted in lotic systems with high current have the potential to produce areas of deposition unaccounted for in the current analysis

4.0 Receptors

4.1 Human Health

Since training activities will occur over open waters, a limited number of human receptors are expected to have access to the area. Furthermore, during training activities, the training area (including a 3.5 mile buffer zone) will be designated as restricted and therefore receptors would not be in the area during training activities.

Although the surface waters at the Great Lakes training location are freshwaters, training activities will not take place near sources of potable water as described in Section 1.3. Therefore, exposure by nearby residents to metals constituents in their drinking water is not a complete exposure pathway and will not be evaluated in this PHA. Surface waters at the Chesapeake Bay training area are estuarine and would not be considered as a drinking water source.

There are no complete exposure pathways involving direct contact with discharged munitions. Since discharged munitions would be in sediments in relatively deep water (>20 feet), recreational swimmers would not come into contact with either munitions or affected sediments. Similarly, leaching of metals from the spent bullets is reasonably expected to be a slow process. Therefore, even if recreators were to swim in the nearby waters, dermal uptake and incidental ingestion of surface water would result in such low rates of exposure that the surface water pathway can reasonably be considered negligible.

Recreational uses, including fishing, are not prohibited in either the Great Lakes or the Chesapeake Bay training areas. It is possible that metals from the discharged bullets could leach and enter the water and sediment. If these compounds are subsequently taken up by edible fish species, it is possible that recreational fishermen could be indirectly exposed to these compounds through ingestion. Based on the conditions at the training sites, the populations most likely to be exposed to metals from discharged munitions in sediments are expected to be recreational fishermen. The receptor category of recreational fisherman represents the maximum or most conservative exposure scenario that would be associated with the training activities. The assumptions used to develop estimates of potential exposure to the recreational fisherman associated with discharge ammunition from training activities are described in Section 6.1. As is discussed in Section 6.1, these assumptions intentionally overestimate potential exposures associated with the training activities.

4.2 Ecological

As indicated in Section 1.3, training exercises were assumed to occur in water depths deeper than 20 ft. Risks associated with training activities at depths shallower that 20 ft. are considered unacceptably high due to the high probability of receptor direct contact with expended ammunition (e.g. direct ingestion of bullet fragments by birds feeding in the sediments). Ecological receptors assessed in this PHA were therefore chosen, primarily,

based their likelihood to inhabit open water environments at depths greater than or equal to 20 ft.

Because of the ecological complexity, it is not possible to directly assess potential impacts to all ecological receptors expected within an area. Therefore, a representative maximally-exposed ecological receptor species or species group were selected as surrogates to represent the larger components of the ecological communities evaluated. Receptor selection was also guided by consideration of the following:

- Are known to occur or are likely to occur in the areas
- Have a particular ecological, economic or aesthetic value
- Are representative of taxonomic groups, life history traits and/or trophic levels in the habitats present for which complete exposure pathways are likely to exist
- Have the greatest potential for exposure
- Can be expected to represent potentially sensitive populations because of toxicological sensitivity or potential exposure magnitude
- Have sufficient ecotoxilogical information available on which to base an evaluation

Fish, water-column invertebrates, and benthic invertebrate species were evaluated based upon those taxonomic groupings for which freshwater and estuarine surface water and sediment screening values have been developed; these groupings and screening values are used in most ecological risk assessments. As such, specific species of aquatic biota, such as lake trout or amphipods, were not chosen as receptor species; aquatic biota will be addressed on a community level via a comparison to surface water and sediment screening values.

Bird and mammal species were selected for evaluation based on the general guidelines presented in USEPA guidance (USEPA, 1991). The following list presents the receptor species identified for evaluation:

- River otter (*Lontra canadensis*) – Common piscivorous mammal found in both freshwater and estuarine environments
- Common loon (*Gavia immer*) – Freshwater avian piscivore declining through much of its range
- Long-tailed duck (*Clangula hyemalis*) – Deep-diving avian piscivore found in freshwater environments
- Osprey (*Pandion haliaetus*)– Well-studied piscivorous raptor found in estuarine environments
- Great blue heron (*Ardea herodias*) – Common piscivorous wading bird found in estuarine environments

Although it is possible that some species of reptiles may be found in the open water environment, individual species of reptiles were not selected for evaluation because of the general lack of available toxicological information for this taxonomic group. Potential risks

to reptiles from exposure through fish ingestion were evaluated using other fauna (birds and mammals) as surrogates. Potential risks from direct exposures to sediment and surface water were evaluated using screening values developed for other taxonomic groups.

5.0 Exposure Assessment

5.1 Sediment

5.1.1 Ammunition Composition

The chemical composition of the expended bullets was used to determine receptor exposure concentrations in sediment, surface water, and fish tissue. The chemical composition was estimated from technical specifications and materials data sheets. The bullets are comprised of four metals: antimony, copper, lead, and zinc. Antimony and lead comprise much of the bullet slug, while the bullet jacket is comprised mostly of copper and zinc. As previously indicated, two types of 7.62 mm bullets can be used. For this investigation, the maximum chemical concentration from either bullet was used in the calculations as a conservative estimate.

5.1.2 Predicted Concentrations

Estimated sediment concentrations for each metal were calculated by multiplying the bullet density (Table 3-1) by the maximum chemical concentration. Sediment concentrations for the Total and Target areas after 5 years are presented in Table 5-1.

5.2 Surface Water Concentration

5.2.1 Surface Water and Sediment Quality Parameters

Water and sediment quality information was obtained from representative sample locations in the Great Lakes and Chesapeake Bay. Great Lakes information was obtained from several sources, including Kemp et al. (1976), EPA's Great Lakes National Program office (general inquiry request for information), and on-line resources (GEMS 2002). Chesapeake Bay information was obtained from the Maryland Department of Natural Resources Chesapeake Bay and Coastal Water Quality on-line database. A sample location (Cedar Point) was selected based on salinity (approximately 13 parts per thousand [ppt]) and depth (greater than 20 ft.) considered representative of an estuarine training location. Descriptions of how specific surface water and sediment quality parameters were used to estimate pore water/surface water concentrations in the geochemical model are provided in Appendix B.

5.2.2 Geochemical Model

The Geochemist's Workbench (Bethke, 2004) was used to estimate pore water concentrations in the Great Lakes and Chesapeake Bay, and is described in further detail in Appendix B.

5.2.3 Predicted Concentrations

Table 5-2 presents the freshwater (maximum for all Great Lakes) and estuarine concentrations of antimony, copper, lead, and zinc in the Total area after 5 years. Also presented in Table 5-2 are the pore water concentrations based on the conservative

assumption that the pore waters would mix with a volume of surface water before there was exposure to aquatic organisms (i.e. it would take 5 cm of surface water to flush pore water from 5 cm of sediment). This assumption produces a 1:1 dilution of surface and pore waters. In actuality the pore water would mix with the entire 20 foot depth, resulting in a dilution of over 100:1.

5.3 Fish Tissue Concentrations

Recreational fisherman and upper-level trophic ecological receptors can be exposed to constituents in discharged ammunition by ingesting fish that have accumulated metals from sediment and water. Bioaccumulation factors (BAFs) were applied to the estimated concentrations in sediment and water to estimate the exposure concentrations in whole-body fish tissue. The BAFs for fish include any bioaccumulation of constituents from sediment or water by plants and invertebrates that are consumed by fish. Although assessment of human health risks typically considers ingestion of just fish fillets instead of whole-body, for this assessment we evaluated human consumption of the entire fish. This approach overestimates the ingestion of COC and thus overestimates risk, because COC typically accumulate to higher levels in fish parts other than the fillets.

5.3.1 Sediment-to-tissue BAFs

Fish tissue concentrations in whole-body fish were estimated by multiplying sediment concentrations by metal-specific sediment-to-fish BAFs obtained from Krantzberg and Boyd (1992) and Pascoe et al. (1996). The BAF values used were based upon the ratio between dry-weight sediment and dry-weight fish tissue. Literature values based upon the ratio between dry-weight sediment and wet-weight fish tissue were converted to a dry-weight basis by dividing the wet-weight BAF by the estimated solids content for fish (25 percent [0.25]; USEPA, 1993). Since a sediment-to-fish BAF for antimony could not be identified in the literature, a BAF of 1.0 was assumed. The sediment-to-fish BAFs are shown in Table 5-3.

5.3.2 Water-to-tissue BAFs

Fish tissue concentrations in whole-body fish were also estimated using water-to-fish bioconcentration factors (BCFs) obtained from USEPA (1999). Since concentrations of metals in both freshwater and marine fish species were used in the derivation of the water BCFs, the values used in this assessment are the same for both the fresh and estuarine surface water. BCF values were converted to BAF values by multiplying the BCF by a food chain multiplier of one for metals (USEPA 1995, 1999). Resulting BAF values were converted to a dry-weight basis by dividing the wet-weight BAF by the estimated solids content for fish (25 percent [0.25]; USEPA 1993). The water-to-fish BAFs are shown in Table 5-3.

5.3.3 Predicted Cconcentrations

The predicted concentrations in fish tissue based on concentrations in sediment and Great Lakes and Chesapeake Bay surface water and media-specific BAFs are shown in Table 5-4. The sediment and water concentrations are those for the total depositional area after 5 years.

5.4 Bird and Mammal Ingestion

Bird and mammal exposures to the ammunition constituents in sediment and surface water were determined by estimating the concentration of each metal in the forage fish as described above. Ingestion of surface water was included when calculating the total exposure. Dietary intakes for each bird and mammals were calculated using the following formula (modified from USEPA [1993]):

$$DI_x = \frac{[(FIR_x)(FC_x) + [(WIR)(WC_x)]}{BW}$$

where:

DI_x	=	Dietary intake for chemical x (mg chemical/kg body weight - day)
FIR	=	Food ingestion rate (kg/day, dry-weight)
FC_{xi}	=	Concentration of chemical x in fish (mg/kg dry-weight)
WIR	=	Water ingestion rate (L/day)
WC_x	=	Concentration of chemical x in water (mg/L)
BW	=	Body weight (kg, wet weight)

The conservative (i.e., high-end) receptor-specific values that were used as input variables to this equation for the screening risk estimates were obtained from relevant scientific literature. Receptor-specific values used as inputs to this equation for the screening risk estimates are provided in Table 5-5. Consistent with the conservative approach used in a screening-level assessment, the minimum body weight and maximum food and water ingestion rates from the scientific literature were used for each receptor. In addition, it was assumed that chemicals are 100 percent bioavailable to the receptor and it was assumed that each receptor spends 100 percent of its time in the Total area.

A list of the key conservative assumptions used in the exposure assessment, their effect on the risk estimate, and more realistic assumptions are presented in Table 5-6.

Table 5-1
Estimated Sediment Concentrations - 5 Years

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Area	Antimony (mg/kg)	Copper (mg/kg)	Lead (mg/kg)	Zinc (mg/kg)
Target	1.16E+00	5.33E+00	1.28E+01	5.90E-01
Total	7.10E-01	3.26E+00	7.83E+00	3.60E-01

Table 5-2

Estimated Pore Water Concentrations - Total Area - 5 Years

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Dilution	Antimony (mg/L)	Copper (mg/L)	Lead (mg/L)	Zinc (mg/L)
Freshwater/Great Lakes [1]				
None	NA [2]	1.03E-04	4.81E-03	2.53E-04
1:1		1.03E-04	4.81E-03	2.53E-04
Estuarine/Chesapeake Bay				
None	NA [2]	4.66E-03	4.04E-03	1.94E-04
1:1		4.66E-03	4.04E-03	1.94E-04

[1] Concentrations are the maximum modeled concentrations of all Great Lakes

[2] Antimony concentrations in pore water could not be reliably estimated

Table 5-3
Sediment and Water Bioaccumulation Factors For Fish
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Chemical	Sediment-Fish BAF (dry weight)		Water-Fish BAF (dry weight)	
	Value	Reference	Value	Reference
Antimony	1.0	Assumed (see text)	800	USEPA 1999
Copper	0.10	Krantzberg and Boyd 1992	2,840	USEPA 1999
Lead	0.070	Krantzberg and Boyd 1992	640	USEPA 1999
Zinc	0.15	Pascoe et al. 1996	2,556	USEPA 1999

Table 5-4
Estimated Fish Tissue Concentrations - Total Area - 5 Years
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Chemical	Sediment (mg/kg wet weight)	Freshwater (mg/kg wet weight)	Estuarine (mg/kg wet weight)
Antimony	1.770E-01	NA [1]	NA [1]
Copper	8.152E-02	7.31E-02	3.31E+00
Lead	1.370E-01	7.70E-01	6.46E-01
Zinc	1.329E-02	1.62E-01	1.24E-01

[1] Antimony concentrations in pore water could not be reliably estimated, and therefore, bioaccumulation to fish tissue from pore was not calculated

Table 5-5
Exposure Parameters for Upper Trophic Level Ecological Receptors
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Receptor	Body Weight (kg)		Water Ingestion Rate (L/day)		Food Ingestion Rate (kg/day - dry)	
	Value	Type/Reference	Value	Type/Reference	Value	Type/Reference
Birds						
Common loon (Freshwater)	3.38	Min. of females in Ontario (McIntyre and Barr 1997)	0.1988	Allometric equation (USEPA 1993)	0.1895	Allometric equation (USEPA 1993)
Long-tailed duck (Freshwater)	0.50	Min. of females (Robertson and Savard 2002)	0.0629	Allometric equation (USEPA 1993)	0.0619	Allometric equation (USEPA 1993)
Osprey (Estuarine)	1.24	Min. of males/females (Dunning 1993)	0.0858	Allometric equation (USEPA 1993)	0.0919	Measured (USEPA 1993)
Great blue heron (Estuarine)	2.10	Min. (Butler 1992)	0.1090	Allometric equation (USEPA 1993)	0.4389	Allometric equation (USEPA 1993)
Mammals						
River Otter (Freshwater and Estuarine)	5.29	Min. for males/females in AL/GA (USEPA 1993)	0.7559	Allometric equation (USEPA 1993)	0.2250	Toweill and Tabor 1982

Table 5-6
Key Realistic Worst-Case Protective Assumptions Incorporated into the Exposure Assessment
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Protective Assumption	Realistic Scenario	Protective Assumption Effect on Risk Estimation
Maximum metal composition in bullets used	Average metal composition in expended bullets	Concentrations over estimated up to 15%
All predictions were based on surface water chemistry that produced the highest concentration of dissolved metals	Average surface chemistry	Concentrations over estimated up to 10 times in most lakes
Bullet was fully exposed and available for dissolution	Only outer portions of copper and zinc in bullet jacket available for dissolution	Concentrations over estimated proportional to bullet competence
Metals from bullets only mix with the bottom 2 inches of water	Metals from bullets would mix with water from the entire water column (i.e., 20 ft.)	Water concentrations over estimated > 20 times based on mixing in the water column
Birds & mammals assumed to have an exclusive fish diet from training area	Significant portion of diet (greater than 50%) based on food items collected outside of depositional area	Risk over estimated proportional to time spent foraging in other areas
Minimum body weight and maximum food and water ingestion rates were used for bird & mammal exposure estimates	Average body weights and ingestion rates	Risk over estimated up to 20%

6.0 Effect Levels

6.1 Human Health

6.1.1 Water Consumption

As described in Section 4.1, there are no direct exposure routes for contact with surface water. However, as a conservative approach, the estimated concentrations of antimony, copper, lead, and zinc in pore water were compared to USEPA Maximum Contaminant Levels (MCLs) for drinking water (USEPA, 2004). This is a conservative comparison since the USEPA MCLs are enforceable standards for public water supplies promulgated under the Safe Drinking Water Act and are designed for the protection of human health. MCLs are set as close as possible (accounting for technical feasibility) to the concentration at which no known or anticipated adverse effects on the health of persons would occur. The health goals are designed to account for a lifetime exposure (70 year lifetime) of an average adult (70 kg) consuming 2 liters of water per day. However since MCLs also consider the technical feasibility of removing the contaminant from the public water supply they are not entirely health-based levels (USEPA 1996). The MCLs are shown in Table 6-1.

6.1.2 Fish Ingestion

Chemical-specific screening values were developed for ingestion of fish tissue based on protection of recreational fishermen. These screening values were based upon the available toxicity information for the constituent metals and exposure factors for recreational fish ingestion. The screening levels in fish tissue were then compared to fish tissue concentrations estimated from sediment and water bioaccumulation, as described in Section 5.3.

Development of Human Health Screening Levels for Fish Tissue

The fish tissue screening levels were derived to be protective of recreational fishermen. The screening levels were derived based on the following expression:

$$SL = \left(\frac{THI \times RfD_{oral} \times BW \times AT_{nc}}{IR_{fish} \times EF \times ED} \right)$$

Where:

SL	=	screening level (mg/kg)
THI	=	target hazard index (unitless)
RfD_{oral}	=	oral reference dose (mg/kg-day)
BW	=	body weight (kg)
AT_{nc}	=	averaging time for noncarcinogenic effects (days)
IR	=	fish ingestion rate (kg/day)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)

The equation requires specific exposure parameters for each receptor (adults and children, ages 1 to 6) and chemical specific toxicity values for each compound (antimony, copper, lead, and zinc).

Exposure Factors

The fish ingestion rate used for the adult recreational fisher screening value was the average fish intake rate for sports-caught fish (4.9 g/day), averaged over 365 days/year (Connelly et al. 1996). This survey was conducted on sports fishermen at Lake Ontario. This fish ingestion rate (4.9 g/day) is consistent with average intake recommended for marine recreational fishing (5.6 g/day for Atlantic Ocean) (USEPA 1997b).

The fish ingestion rate for a child recreational fisherman was averaged from the averages of total fish intakes for children ages 1-2 years (0.37 g/kg-day) and children ages 3 to 5 years (0.32 g/kg-day), as published in USEPA's Child-Specific Exposure Factors Handbook, Table 3-50 (USEPA 2002a). The total fish intake rate was corrected to a sports-caught (recreational) fish intake rate using a factor of 30% provided in Connelly et al., 1996. Table 6-1 presents the exposure factors used in the fish tissue screening level development.

Toxicity Assessment

Toxicity assessment defines the relationship between the magnitude of exposure and possible severity of adverse effects, and weighs the quality of available toxicological evidence. Toxicity assessment generally consists of two steps: hazard identification and dose-response assessment. Dose-response assessment is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose administered or received and the incidence or extent of adverse health effects in the exposed population. Toxicity criteria (e.g., reference doses and slope factors) are derived from the dose-response relationship. Health effects are divided into two broad groups: noncarcinogenic and carcinogenic effects. This division is based on the different mechanisms of action currently associated with each category, and therefore, these differences affect how dose-response is estimated. The target risk levels and toxicity values used in the screening levels were based on noncarcinogenic endpoints, which is consistent with regulatory agency guidance. Attachment A includes toxicity profiles for antimony, copper, lead, and zinc.

Noncarcinogenic health effects are evaluated using the Reference Dose (RfD). USEPA (USEPA 1989) defines the chronic RfD as a dose which is likely to be without appreciable risk of deleterious effects during a lifetime of exposure. Chronic RfDs are specifically developed to be protective for long-term exposure to a compound (7 years to a lifetime), and consider uncertainty in the toxicological database and sensitive receptors. Since the screening levels derived for this assessment are designed to be protective of long term exposure, chronic toxicity values were used when available.

The primary source of toxicity information was the USEPA's Integrated Risk Information System (IRIS). IRIS contains up-to-date toxicity and dose-response information for numerous chemicals that have been verified by USEPA work-groups. RfDs for antimony and zinc were available from IRIS. The RfD for antimony (0.0004 mg/kg-day) is based on a chronic lifetime study with rats. The critical effects identified in the study were reduced

lifespan and changes to the blood (USEPA 2005a). An uncertainty factor of 1,000 was applied to derive the RfD (a factor of 10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and no NOAEL was established). The USEPA lists the overall confidence in the RfD as 'low' (USEPA 2005a).

The oral RfD for zinc (0.3 mg/kg-day) is based on ingestion studies with human volunteers that were designed to establish daily nutritional requirements for zinc. Zinc is an essential trace element crucial to survival, growth, development, and maturation. Thus, insufficient as well as excessive oral intake can cause toxicity and disease. The critical effect observed in the study used to set the RfD was changes in blood chemistry and elevated production of liver enzymes. An uncertainty factor of 3 was used to derive the RfD to account for variability in susceptibility in human populations (USEPA, 2005). The USEPA lists their confidence in the zinc RfD as medium to high (USEPA 2005a).

An RfD for copper was not available from IRIS. Therefore, a noncarcinogenic toxicity value for copper was based on the Minimal Risk Levels (MRLs) developed by the Agency for Toxic Substances and Disease Registry (ATSDR). The ATSDR method for developing MRLs is similar to the USEPA's process for developing RfDs. An MRL is an estimate of the daily human exposure to a substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are derived when ATSDR determines that reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration for a given route of exposure to the substance. ATSDR uses the no observed adverse effect level/uncertainty factor (NOAEL/UF) approach to derive MRLs for substances. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance induced effects.

The ATSDR MRL for copper (0.01 mg/kg-day) is based on gastrointestinal effects from intermediate (up to one year), rather than a chronic exposure period. Since the screening value for recreational fishermen is based on long-term (30 year) exposure, an additional UF of 10 was applied to adjust the intermediate MRL to a chronic exposure basis. Hence, the toxicity value for copper used in the screening level calculations is 0.001 mg/kg-day.

Special Considerations for Lead

The toxicity assessment for lead is different than for the other compounds since an RfD is not available for lead. EPA considered establishing an RfD for inorganic lead in 1985, but concluded that it was inappropriate to develop an RfD for this compound. Therefore, the USEPA regulates lead based on the concentration of lead in blood. The Centers for Disease Control (CDC) identified 10 µg-lead/dL as the blood lead level of concern in children in their 1991 report "Preventing Lead Poisoning in Young Children." Both the CDC and the USEPA have identified children as sensitive receptors for exposure to lead, and therefore the methods used to develop the screening levels were designed to be protective of children. The screening level derived for the adult fisherman is based on potential exposure by a woman of childbearing age, who develops a body burden of lead as a result of fish ingestion over a 30-year period. The fish tissue screening level for lead is based on protection of a fetus that might be carried by a woman consuming fish, under a recreational exposure scenario.

The USEPA has used this information to formulate their stated goal for lead, which states that children (up to 84 months of age) exposed to lead from environmental sources have no more than a 5% probability of exceeding the CDC's level of concern of 10 µg/dL blood-lead level (USEPA 1994a; USEPA 1996). Hence, the screening level has been calculated for an adult woman of child-bearing age based on a fetal blood-lead level of 10 µg/dL and using a biokinetic slope factor relating lead intake from fish ingestion to adult blood-lead level. This approach is based on the USEPA's Adult Lead Model (ALM) (USEPA 1996). It was assumed that 30 percent of the lead in fish tissue was in a bioavailable form (ATSDR 1999).

The child's blood-lead concentration was estimated by using a physiologically-based pharmacokinetic model (the Integrated Exposure Uptake Biokinetic [IEUBK] model). The estimated concentration of lead in fish tissue was used to estimate a risk-based screening value. This is a conservative approach since the IEUBK model also accounts for other exposures to lead, such as lead in soil, drinking water, ambient air, etc. from sources that are not related to the training activities. The IEUBK model assumed that children will consume 1.553 g/day of fish, every day for six years. This ingestion rate was added to the ingestion of lead from other dietary sources in the model. Therefore, the daily intake of lead for children ranged from 8.6 µg/day to 10.1 µg/day.

Human Health Screening Levels for Fish Tissue

The previous sections present the exposure factors and toxicity values used to estimate the chemical-specific screening levels. The fish tissue screening levels were derived to be protective of future recreational adult and child fishermen. Tables 6-3 and 6-4 present the screening values derived for future recreational adult and child fishermen, respectively. Table 6-5 provides documentation of the screening value developed for lead based on the USEPA's ALM methodology. As shown in Figure 6-1, the results of the IEUBK model for a future child recreational fishermen exposed to fish containing 2 mg/kg lead verify that this level of exposure is safe for children. Figure 6-1 shows the mean blood lead concentration for children would be 4.2 µg/dL, which is less than the USEPA level-of-concern concentration (10 µg/dL). Similarly, the figure shows that given this exposure, 3.2% of the population of children with the same exposure would experience blood lead levels greater than 10 µg/dL. This percentage is less than the USEPA's target goal of 5% and therefore will be protective.

6.2 Ecological

6.2.1 Sediment Invertebrates

The source of ecological sediment screening values for sediment invertebrates in the Total area was USEPA Region 3 sediment benchmarks (USEPA 2005b) for freshwater sediment and Effects Range-Low (ER-L) concentrations in Long et al. (1995) for estuarine sediment. USEPA Region 3 freshwater sediment benchmarks, which are the only ones promulgated by EPA, for copper, lead, and zinc are Threshold Effect Concentration (TEC) values from MacDonald et al. (2000). The USEPA Region 3 freshwater sediment benchmark for antimony is the ER-L from Long et al. (1995). TECs and ER-Ls represent concentrations below which adverse effects are not expected to occur (MacDonald et al. 2000). Sediment screening values are shown in Table 6-6.

6.2.2 Surface Water Invertebrates and Fish

The source of freshwater and estuarine ecological surface water screening values for fish and water-column invertebrates in the Total area was chronic criteria in National Ambient Water Quality Criteria (USEPA 2002b) for copper, lead, and zinc. Since no chronic criterion was available for antimony, a freshwater secondary chronic value was obtained from Suter and Tsao (1996), and an estuarine screening value for antimony was obtained from a previous USEPA water quality summary (USEPA 1994b). Freshwater screening values for copper, lead, and zinc require adjustment based on water hardness. Adjustments to these screening values were made using a default, conservative hardness of 100 mg $CaCO_3$/L. Surface water screening values are shown in Table 6-7.

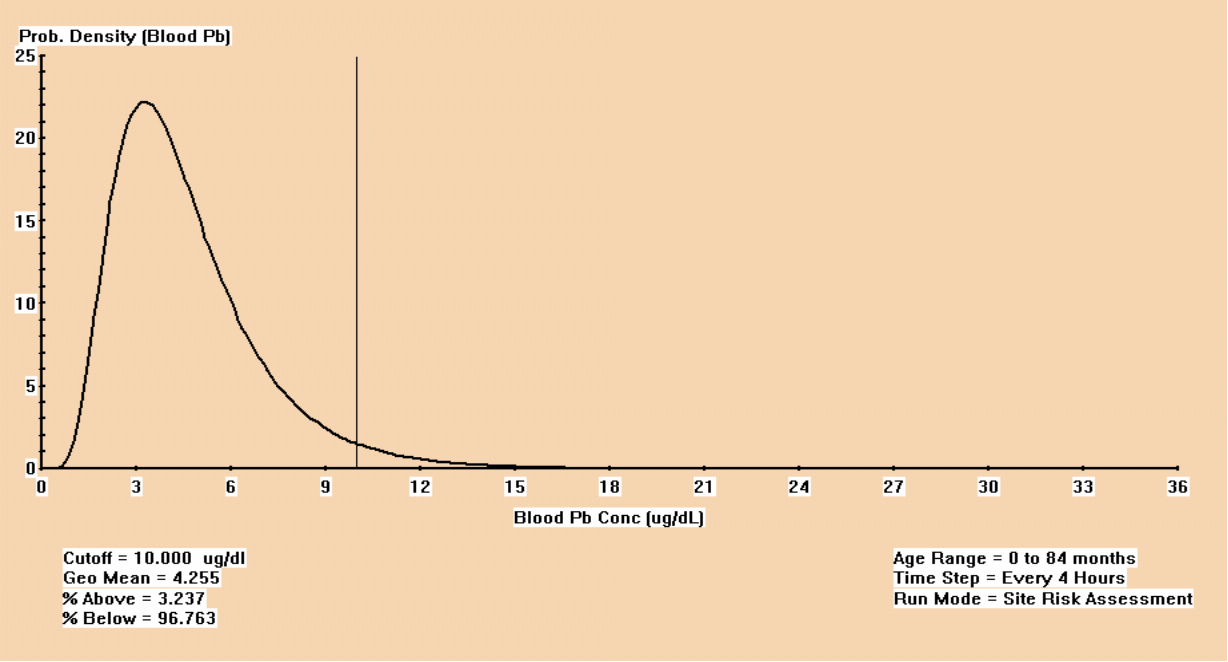
6.2.3 Birds and Mammals

Ingestion screening values for dietary exposures were derived for each mammalian and avian receptor species and metal. Toxicological information from the literature for wildlife species most closely related to the receptor species was used, when available, but was supplemented by laboratory studies of non-wildlife species (e.g., chickens and laboratory rats) when necessary. The ingestion screening values are expressed as milligrams of the chemical per kilogram body weight of the receptor per day (mg/kg-BW/day).

Growth and reproduction were emphasized as assessment endpoints because they are the most ecologically relevant to maintaining viable populations and because they are generally the most studied chronic toxicological endpoints for ecological receptors. If several chronic toxicity studies were available from the literature, the most appropriate study was selected for each receptor species based upon study design, study methodology, study duration, study endpoint, and test species. No Observed Adverse Effect Levels (NOAELs) based on growth and reproduction were used, when available, as the primary screening values. Since a chronic NOAEL was unavailable for antimony, a NOAEL estimate was extrapolated from a chronic Lowest Observed Adverse Effect Level (LOAELs) using an uncertainty factor of 10. Ingestion screening values for mammals and birds are summarized in Table 6-8.

A list of the key conservative assumptions used in the effects assessment, their effect on the risk estimate, and more realistic assumptions are presented in Table 6-9.

FIGURE 6-1
Calculation of Blood-Lead Levels for Child Recreational Fish Consumption
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises



****
ADDITIONAL MODEL INPUTS:
****
The time step used in this model run: 1 - Every 4 Hours (6 times a day).

******Air******

Indoor Air Pb Concentration: 30.000 percent of outdoor.
Other Air Parameters:

Age	Time Outdoors (hours)	Ventilation Rate (m^3/day)	Lung Absorption (%)	Outdoor Air Pb Conc ug Pb/m^3
.5-1	1.000	2.000	32.000	0.100
1-2	2.000	3.000	32.000	0.100
2-3	3.000	5.000	32.000	0.100
3-4	4.000	5.000	32.000	0.100
4-5	4.000	5.000	32.000	0.100
5-6	4.000	7.000	32.000	0.100
6-7	4.000	7.000	32.000	0.100

FIGURE 6-1
Calculation of Blood-Lead Levels for Child Recreational Fish Consumption
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

******Diet******

Fish and other dietary sources:

Age	Diet Intake(ug/day)
.5-1	8.638
1-2	8.888
2-3	9.598
3-4	9.348
4-5	9.118
5-6	9.448
6-7	10.108

Fish tissue concentration: 2 ug Pb/g

******Drinking Water******

Water Consumption:

Age	Water (L/day)
.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580
6-7	0.590

Drinking Water Concentration: 4.000 ug Pb/L

******Soil& Dust******

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
.5-1	400.000	50.000
1-2	400.000	50.000
2-3	400.000	50.000
3-4	400.000	50.000
4-5	400.000	50.000
5-6	400.000	50.000
6-7	400.000	50.000

******Maternal Contribution: Infant Model******

Maternal Blood Concentration: 2.500 ug Pb/dL

****

CALCULATED BLOOD LEAD AND LEAD UPTAKES:

****

Year	Air (ug/dL)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)
5-1	0.021	3.894	0.000	0.361	4.770	9.046	4.9
1-2	0.034	3.982	0.000	0.896	7.530	12.442	5.2
2-3	0.062	4.355	0.000	0.944	7.627	12.988	4.8
3-4	0.067	4.301	0.000	0.975	7.733	13.076	4.6
4-5	0.067	4.287	0.000	1.034	5.854	11.243	3.9
5-6	0.093	4.480	0.000	1.100	5.313	10.986	3.5
6-7	0.093	4.812	0.000	1.124	5.038	11.068	3.2

Table 6-1
Risk Screening Concentrations - Human Health - Maximum Contaminant Levels for Drinking Water
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Chemical	Screening Value (µg/L)	Reference
Antimony	6.00E+00	USEPA 2004
Copper	1.30E+03	USEPA 2004
Lead	1.50E+01	USEPA 2004
Zinc	2.00E+03	USEPA 2004

Table 6-2
Exposure Factors Used for Screening Levels - Adult and Child Recreational Fish Consumption
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Exposure Parameter	Units	Adult Recreational Fisherman		Child Recreational Fisherman	
		Value	Reference	Value	Reference
THI - Target Hazard Index	--	1		1	
C - Fish Tissue Concentration Corresponding to Target Risk	mg/kg	Calculated		Calculated	
IR - Fish Ingestion Rate	g/day	4.9	(1)	1.554	(3)
BW - Body Weight	kg	70	USEPA 1991b	15	USEPA 1991b
EF - Exposure Frequency	days/year	365	Prof. Judgment	365	Prof. Judgment
ED - Exposure Duration	year	30	(2)	6	(4)
AT_n - Averaging time - noncarcinogenic	day	10,950	USEPA 1989	2,190	USEPA 1989

(1) The fish ingestion rate for the adult recreational fisher is the average fish intake rate for sports-caught fish (4.9 g/day), averaged over 365 days/year, presented in Connelly et al., 1996.

(2) Professional judgment assuming the same exposure duration as an adult resident (USEPA 1991b).

(3) The fish ingestion rate for the child recreational fisher is based on the average daily intake rates for all fish consumed by children ages 1-2 and 3-5, presented in child-specific exposure factors developed by USEPA (USEPA 2002a). The child fish intake rates were then corrected by a factor of 30 percent of total fish intake being due to sports-caught fish, presented in Connelly et al., 1996.

(4) Professional judgment assuming the same exposure duration as a child resident (USEPA 1991b).

Table 6-3
Calculation of Risk-Based Screening Values for Adult Recreational Fish Consumption
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Calculations:

$$\text{Intake (mg/day) Based on THI} = \frac{\text{Oral Reference Dose (mg/kg-day)}}{\text{BW (kg)}}$$

$$\text{Fish Tissue Concentration (mg/kg) Corresponding to THI} = \frac{\text{I (mg/day)} \times AT_n \text{ (days)}}{\text{IR (g/day)} \times 0.001 \text{ (kg/g)} \times \text{EF (day/year)} \times \text{ED (year)}}$$

Exposure Parameter	Value	Units
TR - Target Risk Level	1.00E-06	--
THI - Target Hazard Index	1	--
C - Fish Tissue Concentration Corresponding to Target Risk	Calculated	mg/kg
I - Intake	Calculated	mg/day
IR - Fish Ingestion Rate	4.9	g/day
BW - Body Weight	70	kg
EF - Exposure Frequency	365	days/year
ED - Exposure Duration	30	year
AT_n - Averaging time - noncarcinogenic	10950	day
AT_c - Averaging time - carcinogenic	25550	day

Chemical	Oral Cancer Slope Factor (kg/day-mg)	Oral RfD (mg/kg-day)	Source	Intake Based on Target Hazard Index (mg/day)	Fish Tissue Concentration Corresponding to Target Hazard Index (mg/kg)
Antimony	NA	0.0004	IRIS	0.028	5.7
Copper	NA	0.001	ATSDR MRL[a]	0.070	14
Lead	NA	NA	NA	NA	2.0
Zinc	NA	0.3	IRIS	21	4286

[a] Extrapolated from an intermediate duration MRL of 0.001 mg/kg-day, multiplied by 10-fold uncertainty factor to convert from less than lifetime to lifetime

ATSDR - Agency for Toxic Substances and Disease Registry.
IRIS - Integrated Risk Information System Database (USEPA 2005a).
MRL - Minimal Risk Level.
NA - Not available or not applicable.
The fish ingestion rate for the adult recreational fisher is the average fish intake rate for sports-caught fish (4.9 g/day), averaged over 365 days/year, presented in Connelly et al. 1996.

Table 6-4
Calculation of Risk-Based Screening Values for Child Recreational Fish Consumption
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Calculations:

$$\text{Intake (mg/day) Based on THI} = \frac{\text{Oral Reference Dose (mg/kg-day)}}{\text{BW (kg)}}$$

$$\text{Fish Tissue Concentration (mg/kg) Corresponding to THI} = \frac{\text{I (mg/day)} \times AT_n \text{ (days)}}{\text{IR (g/day)} \times 0.001 \text{ (kg/g)} \times \text{EF (day/year)} \times \text{ED (year)}}$$

Exposure Parameter	Value	Units
TR - Target Risk Level	1.00E-06	--
THI - Target Hazard Index	1	--
C - Fish Tissue Concentration Corresponding to Target Risk	Calculated	mg/kg
I - Intake	Calculated	mg/day
IR - Fish Ingestion Rate	1.554	g/day
BW - Body Weight	15	kg
EF - Exposure Frequency	365	days/year
ED - Exposure Duration	6	year
AT_n - Averaging time - noncarcinogeni	2190	day
AT_c - Averaging time - carcinogenic	25550	day

Chemical	Oral Cancer Slope Factor (kg/day-mg)	Oral RfD (mg/kg-day)	Source	Intake Based on Target Hazard Index (mg/day)	Fish Tissue Concentration Corresponding to Target Hazard Index (mg/kg)
Antimony	NA	0.0004	IRIS	0.0060	3.9
Copper	NA	0.001	ATSDR MRL[a]	0.015	9.7
Lead	NA	NA	NA	NA	2.0
Zinc	NA	0.3	IRIS	4.5	2896

[a] Extrapolated from an intermediate duration MRL of 0.001 mg/kg-day, multiplied by 10-fold uncertainty factor to convert from less than lifetime to lifetime

ATSDR - Agency for Toxic Substances and Disease Registry.

MRL - Minimal Risk Level.

NA - Not available or not applicable.

The fish ingestion rate for the child recreational fisher is based on the average daily intake rates for all fish consumed by children ages 1-2 and 3-5, presented in child-specific exposure factors developed by USEPA (USEPA 2002a). The child fish intake rates were then corrected by a factor of 30 percent of total fish intake being due to sports-caught fish, presented in Connelly et al. 1996.

Table 6-5

Calculation of Risk-Based Lead Screening Value for Adult Recreational Fish Consumption: Output from U.S. EPA Adult Lead Model Adapted for Fish Consumption

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Exposure Variable	PRG Equation [1] 1*	PRG Equation [1] 2**	Description of Exposure Variable	Units	Values for Non-Residential Exposure Scenario Using Equation 1 GSDi = Hom	Values for Non-Residential Exposure Scenario Using Equation 1 GSDi = Het
$PbB_{fetal,\ 0.95}$	X	X	95^{th} percentile PbB in fetus	ug/dL	10	10
$R_{fetal/maternal}$	X	X	Fetal/maternal PbB ratio	--	0.9	0.9
BKSF	X	X	Biokinetic Slope Factor	ug/dL per ug/day	0.4	0.4
GSD_i	X	X	Geometric standard deviation PbB	--	2.0	2.2
PbB_0	X	X	Baseline PbB	ug/dL	1.4	2.0
IR_S	X		Fish ingestion rate	g/day	4.900	4.900
IR_{S+D}		X	Total ingestion rate of outdoor soil and indoor dust	g/day	--	--
W_S		X	Weighting factor; fraction of IR_{S+D} ingested as outdoor soil	--	--	--
K_{SD}		X	Mass fraction of soil in dust	--	--	--
$AF_{S,\ D}$	X	X	Absorption fraction	--	0.30	0.30
$EF_{S,\ D}$	X	X	Exposure frequency	days/yr	365	365
$AT_{S,\ D}$	X	X	Averaging time	days/yr	365	365
PRG			Preliminary Remediation Goal	mg/kg	4	2

[1] Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).
When $IR_S = IR_{S+D}$ and $W_S = 1.0$, the equations yield the same PRG.

*Equation 1, based on Eq. 4 in USEPA (1996).

$$PRG = \frac{([PbB_{95}fetal/(R*(GSD_i^{1.645})])-PbB_0)*AT_{S,D}}{BKSF*(IR_{S+D}*AF_{S,D}*EF_{S,D})}$$

Adult Lead Model, Version Date 05/19/03. U.S. EPA Technical Review Workgroup for Lead, Adult Lead Committee.

Table 6-6
Risk Screening Concentrations - Ecological - Sediment
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

	Freshwater		Estuarine	
Chemical	Screening Value (mg/kg)	Type/Reference	Screening Value (mg/kg)	Type/Reference
Antimony	2.00E+00	ER-L (USEPA 2005b)	2.00E+00	ER-L (Long et al. 1995)
Copper	3.20E+01	TEC (USEPA 2005b)	3.40E+01	ER-L (Long et al. 1995)
Lead	3.60E+01	TEC (USEPA 2005b)	4.67E+01	ER-L (Long et al. 1995)
Zinc	1.21E+02	TEC (USEPA 2005b)	1.50E+02	ER-L (Long et al. 1995)

Table 6-7
Risk Screening Concentrations - Ecological - Surface Water
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

	Freshwater		Estuarine	
Chemical	Screening Value (μg/L)	Type/Reference	Screening Value (μg/L)	Type/Reference
Antimony	3.00E+01	SCV (Suter and Tsao 1996)	5.00E+02	Proposed CCC (USEPA 1994a)
Copper	8.96E+00	CCC (USEPA 2002b)	3.10E+00	CCC (USEPA 2002b)
Lead	2.52E+00	CCC (USEPA 2002b)	8.10E+00	CCC (USEPA 2002b)
Zinc	1.13E+02	CCC (USEPA 2002b)	8.10E+01	CCC (USEPA 2002b)

Table 6-8
Risk Screening Doses - Ecological - Mammals
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Chemical	Test Organism	Body Weight (kg)	Duration	Exposure Route	Effect/Endpoint	Screening Value	Type/Reference
Mammals							
Antimony	mouse	0.03	lifetime	oral in water	lifespan/longevity	0.125	NOAEL (Sample et al. 1996)
Copper	mink	1.00	357 days	oral in diet	reproduction	11.7	NOAEL (Sample et al. 1996)
Lead	rat	0.35	3 generations	oral in diet	reproduction	8.00	NOAEL (Sample et al. 1996)
Zinc	mink	1.00	25 weeks	oral	reproduction	20.8	NOAEL (ATSDR 1994)
Birds							
Antimony	Screening value not available						
Copper	chicken (chicks)	0.534	10 weeks	oral in diet	growth/survival	47.0	NOAEL (Sample et al. 1996)
Lead (Canada Goose)	Japanese quail	0.15	12 weeks	oral in diet	reproduction	1.13	NOAEL (Sample et al. 1996)
Lead (Herons and Osprey)	American kestrel	0.13	7 months	oral in diet	reproduction	3.85	NOAEL (Sample et al. 1996)
Zinc	chicken	1.94	44 weeks	oral in diet	reproduction	14.5	NOAEL (Sample et al. 1996)

Table 6-9
Key Realistic Worst-Case Protective Assumptions Incorporated into the Effects Assessment
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Protective Assumption	Realistic Scenario	Protective Assumption Effect on Risk Estimation
Pore water concentrations were compared to drinking water MCLs	Training exercises will not be conducted near potable water intakes; therefore, ingestion of pore water is not expected to occur	Risks estimates are provided that are unnecessary
Bird and mammal screening values based on forms of metal (such as salts) that have high water solubility and high bioavailability	Actual bird and mammal screening values an order of magnitude higher	Risks overestimated up to 10 times
Direct exposure to aquatic organisms evaluated with chronic criteria	Aquatic organisms spend significant portion of time outside of depositional area and therefore effects are best determined using acute criteria	Risks overestimated up to 10 times

7.0 Risk Characterization

The risk characterization is the final step in a PHA. In this step, the estimated exposure concentrations or doses (birds and mammals) are compared with the corresponding screening values to derive screening risk estimates. The screening risk estimates are intended are used to answer one of three general questions for each risk scenario evaluated:

- Are significant elevated risks predicted using "realistic worst case" assumptions?
- Are no elevated risks predicted even with "realistic worst case" assumptions?
- Is there an unacceptale level of uncertain, such that unanticipated occurances result in elevated risks?

Based on the outcome of this evaluation, recommendations will be made about the need for additional investigations, including the initiation of a Baseline Risk Assessment (BRA), if necessary.

Screening risk estimates are determined using the screening risk quotient method. Screening risk quotients are calculated by dividing the constituent concentration in the medium being evaluated by the corresponding medium-specific screening value or by dividing the exposure dose by the corresponding ingestion screening value.

Screening risk quotients exceeding one indicate the potential for risk because the constituent concentration or dose (exposure) exceeds the screening value (effect). However, screening values and exposure estimates are derived using intentionally conservative assumptions such that risk quotients greater than or equal to one do not necessarily indicate that risks are present or impacts are occurring. Rather, it identifies constituent-pathway-receptor combinations requiring further evaluation. Risk quotients that are less than one indicate that risks are very unlikely, enabling a conclusion of no significant elevated risk to be reached with high confidence.

7.1 Human Health

7.1.1 Water Consumption

Freshwater and estuarine pore water concentrations assuming a 1:1 dilution in the Total area after five years are compared to human health drinking water MCLs in Figure 7-1. Freshwater and estuarine concentrations of copper, lead, and zinc did not exceed their respective MCLs, and risk from these metals to human health from water consumptions are considered negligible. The maximum screening quotient was 0.16 for lead in freshwater. Freshwater and estuarine concentrations of antimony could not be reliably estimated and, therefore, a comparison to screening values was not performed.

7.1.2 Fish Consumption

The maximum fish tissue concentrations in freshwater and estuarine systems (based on water or sediment bioaccumulation) in the Total area after 5 years are compared to fish tissue concentrations protective of children from recreational fishing in Figure 7-2. Since freshwater and estuarine concentrations of antimony could not be reliably estimated, fish tissue concentrations based on only sediment bioaccumulation were available. Maximum fish tissue concentrations of antimony, copper, lead, and zinc did not exceed protective concentrations, and risk from these metals to human health from fish ingestions are considered negligible. The maximum screening quotient was 0.39 for lead in freshwater.

7.2 Ecological

7.2.1 Sediment Invetebrates

Sediment concentrations in the Target and Total areas after five years are compared to freshwater and estuarine ecological screening values in Figure 7-3. Sediment concentrations of antimony, copper, lead, and zinc did not exceed freshwater or estuarine screening values, and, therefore, risks from these metals to sediment invertebrates are considered negligible. The maximum screening quotient was 0.58 for antimony in the Target area.

7.2.2 Surface Water Invertebrates and Fish

Freshwater and estuarine concentrations in the Total depositional areas after five years and assuming a 1:1 dilution are compared to freshwater and estuarine ecological screening values in Figure 7-4. Freshwater and estuarine concentrations of copper, lead, and zinc did not exceed their respective screening values, and risks from these metals to fish and water column invertebrates are considered negligible. The maximum screening quotient was 0.96 for lead in freshwater. Freshwater and estuarine concentrations of antimony could not be reliably estimated and, therefore, a comparison to screening values was not performed.

7.2.3 Mammals and Birds

Screening quotients based upon maximum exposure doses for each freshwater and estuarine upper trophic level ecological receptor in the Total area after five years are shown in Figures 7-5 and 7-6, respectively. Each screening quotient shown in Figures 7-5 and 7-6 are based on the maximum fish tissue concentration (based on water or sediment bioaccumulation). Based upon a comparison to NOAELs, screening quotients for copper, lead, and zinc were below one and risks to mammals and birds are considered negligible. The maximum screening quotient was 0.14 for lead and the osprey. As indicated previously, concentrations of antimony could not be reliably estimated and, therefore, a comparison to screening values was not performed.

7.3 Additional Evaluation of Marine Environments

Subsequent to preparation of the PHA, risks in marine sediments from live gunnery training were qualitatively evaluated. Risks in the marine water column were not considered significant because analyses for the other settings (i.e. lakes, estuaries, and rivers)

conclusively showed no elevated risk in water, with predicted concentrations in sediments closer to criteria. Thus if there was any indication of elevated risk in marine settings, it would appear in the sediments before the water column. Risks in the marine sediment were considered similar to other environments and with no indication of elevated risk.

7.4 Summary of Results

In summary, sediment, surface water, and fish tissue concentrations of antimony, copper, lead, and zinc did not exceed human health and ecological screening values.

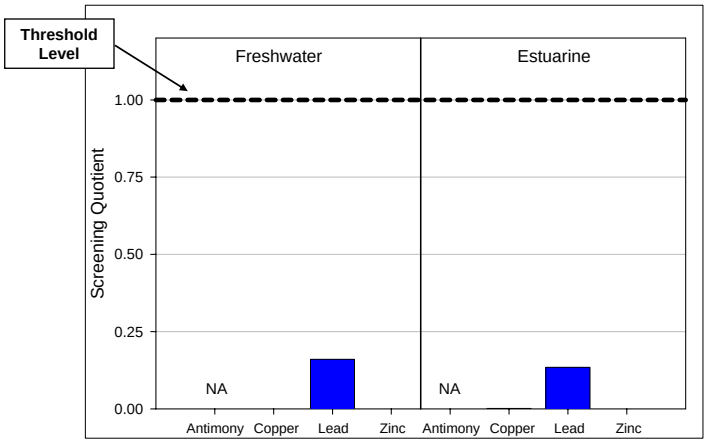


Figure 7-1. Screening Quotients for Maximum Pore Water Concentrations Assuming 1:1 Dilution over the Total Training Area and Human Health MCLs

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

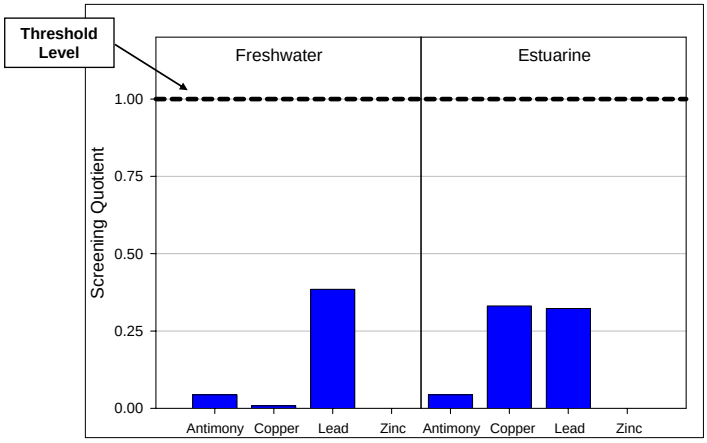


Figure 7-2. Screening Quotients for Fish Tissue Concentrations Protective of Human Health (Child) Based on Maximum of Pore Water or Sediment over the Total Training Area

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

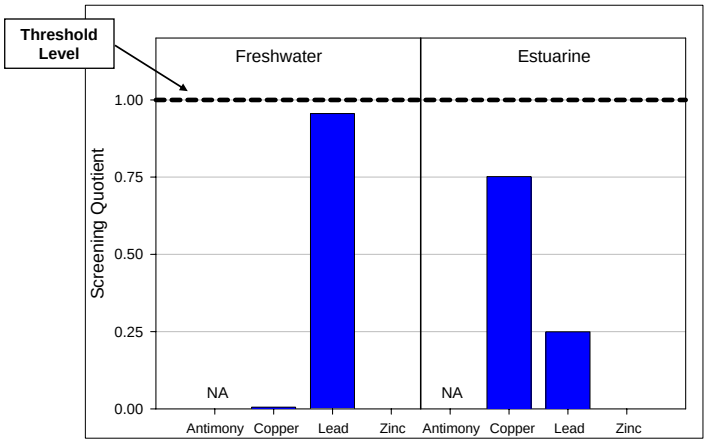


Figure 7-3. Screening Quotients for Estimated Pore Water Concentrations Assuming 1:1 Dilution in the Total Training and Surface Water Criteria

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

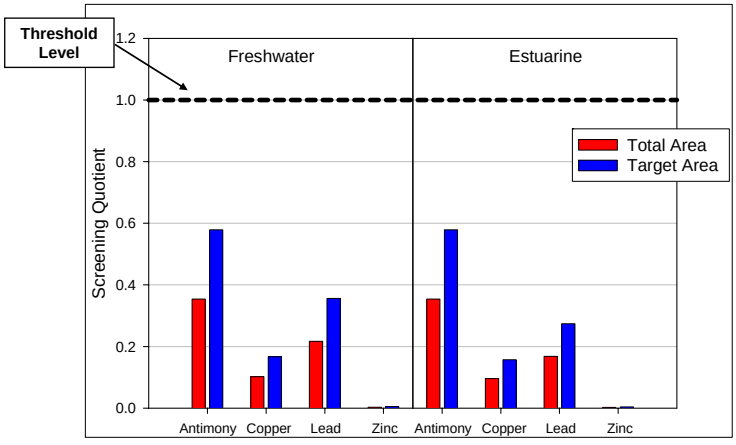


Figure 7-4. Screening Quotients for Estimated Sediment Concentrations in the Total and Target Areas and Sediment Criteria

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

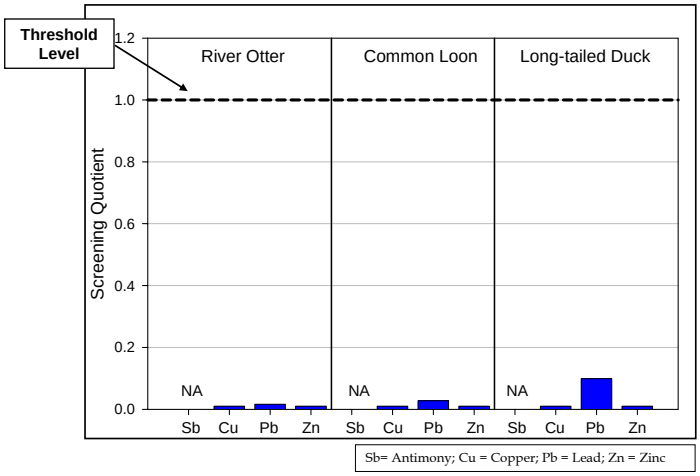


Figure 7-5. Screening Quotients for Fish Tissue Concentrations Based on Maximum of Pore Water (No Dilution) or Sediment in Freshwater over the Total Training Area

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

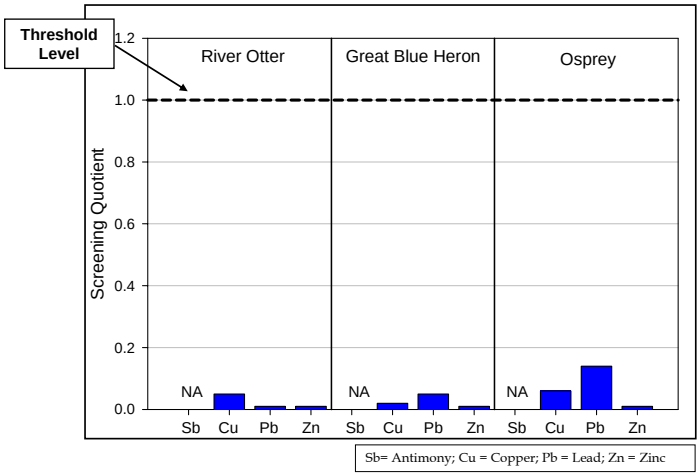


Figure 7-6. Screening Quotients for Fish Tissue Concentrations Based on Maximum of Pore Water (No Dilution) or Sediment in Estuarine Waters over the Total Training Area

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

8.0 Assumptions and Uncertainties

Uncertainties are present in all risk assessments because of the limitations of the available data and the need to make certain assumptions and extrapolations based upon incomplete information. As stated in Sections 1.1 and 1.3, the overall approach for this PHA used "realistic worst-case" assumptions, which were intended to be conservatively protective of human health and ecological receptors as well as provide for reasonable limitations on training activities. The uncertainties in this PHA as a result of these assumptions are listed below for each section.

8.1 Conceptual Model

- The conceptual model for the lotic/riverine system is the same as that for the Great Lakes/freshwater system. Specific receptors, as well as sediment, surface water, and tissue concentrations, were considered similar, such that risk conclusions and assumptions for the Great Lakes/freshwater system also apply to slow-moving riverine systems. Training activities conducted in areas of high current and high rates of deposition immediately downstream have the potential to produce areas of deposition unaccounted for in the current analysis. Therefore, areas of high current and high rates of deposition immediately downstream should be avoided for training exercises. Risks estimates presented in the PHA could be underestimated if training activities are conducted in lotic areas with high current and high rates of deposition immediately downstream.

8.2 Source

- For this evaluation, the discharged ammunition was conservatively modeled as sediment (i.e., the weight and chemical composition of the discharged bullets was assumed to be sediment with an equal weight and chemical composition available to the aquatic system). This assumption results in an over-estimation of risk because metals will dissolve out of the bullets and adsorb to sediments at much lower levels.

- Existing or background contamination from previous activities is not considered, and therefore, current risks can not be evaluated. The risk evaluation only considers future risks based on use of hypothetical test firing areas. Training exercises that take place in areas with existing levels of contamination with the potential for cumulative impacts with expended ammunition are not considered in this PHA. Risks estimates presented in the PHA are underestimated if contamination currently exists in training areas, although risks would not be entirely attributable to USCG training activities. As long as the existing sediment concentrations are not already near or over screening levels used in this PHA, the addition of metals from USCG training operations will not result in elevated risk. Even if existing concentrations are elevated above screening concentrations, USCG operations should not result in a measurable elevation of risk.

- Only bullets will be discharged to the water. Other components of the ammunition (e.g.., cartridges) were not considered in this risk evaluation. While it is expected that other components of the ammunition may be incidentally discharged on an infrequent basis, the risks associated with these other components is expected to be negligible. However, risks estimates presented in the PHA are underestimated if there is excessive discharge of other ammunition components.
- There are two types of 7.62 mm bullets. The bullet with the greatest mass (149 grains or 9.7 grams) was selected for modeling purposes as a conservative assumption. Risks estimates presented in the PHA are overestimated if the other bullet type is predominantly used in training exercises.
- Bullets from other weapons were not considered in this PHA. Although a rifle and carbine may be used in training activities, they bullet size used for the weapon or the annual expenditure of bullets is less than that with the 7.62 mm bullet. Therefore, risks presented in this PHA are overestimated if other weapons are used for training exercises.
- The depositional area was calculated using several assumptions, including the assumption that the quantity of ammunition discharged was evenly divided among 12 areas, 30% of the bullets were expected to be deposited within 20 yards of the target, and there was surface drift of up to 0.25 knots (see Section 3.0 for all assumptions included in the calculation of the depositional area). Risk estimates may be underestimated if there are significant deviations from these assumptions.
- To account for cumulative deposition over the five year training duration, annual expenditures were added together to calculate the Total area bullet density. It was assumed that the Target area would not be at the same position, but would be within the previous year's Total depositional area. Sediment concentrations are the same for both freshwater and estuarine systems. While these assumptions are considered conservative and risk estimates presented in the PHA are overestimated based on these assumptions, risk estimates may be underestimated if Target areas overlap over a five year period.
- The additional chemicals associated with tracer rounds were not included in the risk estimates presented in this PHA (i.e., all discharged ammunition was considered to be normal service ammunition). Tracer rounds include chemicals in addition to those metals evaluated in this PHA. However, these chemicals are not toxic in the aquatic environment and quantities are very small relative to the quantities of metals present in the bullet and cartridge. Also, the chemicals typically evaporate before contact with surface water. There is no anticipated elevated risk associated with normal use of tracer rounds (4 rounds of normal service ammunition discharged per 1 tracer round and were not evaluated in this PHA.

8.3 Receptors

- Reptiles were not evaluated quantitatively in the PHA, and were evaluated using other fauna (birds and mammals) as surrogates due to the general lack of taxon-specific toxicological data. This represents an uncertainty in the assessment. It was also assumed

that reptiles were not exposed to significantly higher concentrations of chemicals and were not more sensitive to chemicals than the other receptor species evaluated. This assumption was a source of uncertainty in the ERA. In addition, there is some uncertainty associated with the use of specific receptor species to represent larger groups of organisms (e.g., guilds).

8.4 Exposure Assessment

- Training activities will not be conducted in the vicinity of the most senstive areas, such as potable water intakes, national wildlife refuges, and national parks. The location of these areas should be considered prior to initiation of training exercises, and avoided, if possible. The risks estimates presented in the PHA could be underestimated if training activities are conducted in close proximity to these areas.
- Five years of training activity are evaluated. Risks associated with training activities that extend beyond this five-year period are not evaluated in this PHA.
- The model estimation assumes that the bed sediment pore water is the same as the lake and bay water. However, the pore water will include microbes that can significantly alter not only the water chemistry to which the metals are exposed but also both the rate at which the metals chemically react and the minerals formed by the chemical reaction. The model assumes, at this point, that the chemical reactions are totally abiotic (no microbial activity). Risk estimates based on this assumption are therefore overestimated.
- Effectively, modeled dissolved metals concentrations estimates assume a constant volume of water surrounding the metals (pore water). Each of the metals are also assumed to be completely exposed (i.e., no compartmentalization of bullet and slug), whereas an unknown but significant portion of the munitions will not be exposed until the more exposed portions are almost totally reacted. Risk estimates based on this assumption are therefore overestimated.
- Chemical concentrations in fish were modeled from predicted concentrations in sediment and surface water. The use of generic, literature-derived exposure models and bioaccumulation factors introduces some uncertainty into the resulting estimates. The values selected were intended to provide a conservative estimate of potential food web exposure concentrations. Risk estimates based on these modeled concentrations are therefore overestimated.
- BAFs for fish tissue are based on whole-body concentrations whereas human health exposure is typically for the fillet only. Risk estimates based on estimated whole body fish tissue concentrations are therefore overestimated if only the fillet is ingested.
- Bird and mammal receptors were assumed to have an exclusive diet of fish from the training area. For some of the receptors, other prey items, such as invertebrates or plant material, may also be ingested. Since training exercises were assumed to be conducted at a minimum of 20 ft, these other prey items, if present in the depositional area, are unlikely to be collected by the bird and mammal receptors evaluated in this PHA. The risks associated with ingestion of other prey items are therefore considered negligible

and were not evaluated in this PHA. Risks estimates based on exclusive diets of fish from the depositional area are therefore overestimated.

- Since a sediment-to-fish BAF for antimony could not be identified in the literature, a BAF of 1.0 was assumed. This value is likely an overestimate of antimony bioaccumulation in aquatic systems (USEPA 2000), and risks presented in the PHA based on antimony-levels in fish are overestimated.
- Metals in surface water, sediment, and fish tissue were assumed to be 100 percent bioavailable to ecological receptors. Risk estimates based on these assumptions are overestimated.
- Area use factors for ecological receptors were assumed to equal 1.0. This is a conservative assumption since a significant percentage of each bird and mammal species time could be spent foraging in other areas or areas where chemical concentrations are expected to be significantly lower. Risk estimates based on this assumption are overestimated.

8.5 Effect Levels

- Data on the toxicity of many chemicals to the ecological receptor species were sparse or lacking, requiring the extrapolation of data from other wildlife species or from laboratory studies with non-wildlife species. This is a typical limitation and extrapolation for ecological risk assessments because so few wildlife species have been tested directly for most chemicals. The uncertainties associated with toxicity extrapolation were minimized through the selection of the most appropriate test species for which suitable toxicity data were available. The factors considered in selecting a test species to represent a receptor species included taxonomic relatedness, trophic level, foraging method, and similarity of diet. Risk estimates based on these assumptions may be overestimated or underestimated.
- Effects-Range Low (ERL) values are used in this PHA, but, as noted by O'Connor (2004), these values do not represent a threshold concentration in sediment at which the probability of toxicity shows an abrupt increase. Similarly, there is no basis for assuming that multiple concentrations above an ERL increase the probability of toxicity.
- Most of the toxicological studies on which the ingestion screening values were based on forms of the metal (such as salts) that have high water solubility and high bioavailability to receptors. These highly bioavailable forms are expected to compose only a fraction of the total metal concentration. This is likely to result in an overestimation of potential risks for these chemicals.
- A mammal screening value for antimony was extrapolated from a LOAEL using an uncertainty factor of ten. This approach is likely to be conservative since Dourson and Stara (1983) determined that 96 percent of the chemicals included in a data review had LOAEL/NOAEL ratios of five or less. The use of an uncertainty factor of 10, although potentially conservative, also serves to counter some of the uncertainty associated with interspecies extrapolations, for which a specific uncertainty factor was not used.

- A bird screening value for antimony was not available and risk to birds from this metal could be not be determined, but are likely to be negligible. Antimony is not considered a bioaccumulative chemical (USEPA, 2000), and, therefore, sources of antimony to avian receptors would only be from ingested surface water and sediment, which were small percentages of the modeled exposures.

8.6 Risk Characterization

- Information on the ecotoxilogical effects of chemical interactions is generally lacking, which required (as is standard for ecological risk assessments) that the chemicals be evaluated on a compound-by-compound basis during the comparison to screening values. This could result in an underestimation of risk (if there are additive or synergistic effects among chemicals) or an overestimation of risks (if there are antagonistic effects among chemicals).

9.0 Summary

The overall objective of this PHA was to evaluate potential risks to human and ecological receptors associated with expended small caliber munitions from USCG live gunnery training. This evaluation intended to answer one of three general questions for each risk scenario evaluated:

- Are significant elevated risks predicted using "realistic worst case" assumptions?
- Are no elevated risks predicted even with "realistic worst case" assumptions?
- Is there an unacceptable level of uncertain, such that unanticipated occurances result in elevated risks?

Based on the results of this evaluation, proposed training will result in no elevated risks for a freshwater system such as the Great Lakes, estuarine system such as the Chesapeake Bay, or generic marine environments using "realistic worst case" assumptions, and further investigation is not recommended. If typical rather than worst case assumptions were used the predicted risk would be even less.

10.0 References

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Appendices

Appendix A: Toxicity Profiles

A.1 Ecological

Antimony

Antimony is a naturally occurring metalloid element (displaying both metallic and nonmetallic properties) existing in valence states of 3 and 5 (Budavari, 1989; ATSDR, 1990). In waterways, antimony is generally associated with particular matter (ATSDR, 1990). Trivalent antimony compounds are the most significantly bioavailable species (ATSDR, 1990). However, studies on fish have suggested that antimony bioaccumulation is not very likely (ATSDR, 1990; Callahan et al., 1979). In a 28-day exposure, bluegill did not accumulate antimony above concentrations in control fish and the bioconcentration factor was less than 1.0 (USEPA, 1980).

A screening value for mammals was based on a study reviewed in Sample et al. (1996). A one year study conducted on the effects of antimony on the growth, survival, and tissue levels in mice indicated a chronic oral toxicity dose of 5 ppm (Schroeder et al., 1968). This dose was converted to 1.25 mg/kg/day and considered a chronic LOAEL because median life span was reduced among female mice exposed to the 5 ppm dose level. A chronic NOAEL of 0.125 mg/kg/day was estimated by multiplying the chronic LOAEL by an uncertainty factor of 0.1. A reliable screening value for birds could not be identified in the literature.

Copper

Copper occurs in natural waters primarily as the divalent cupric ion in free and complexed forms (USEPA, 1985). Copper may be deposited in sediments when adsorbed to organic matter or precipitated with hydroxides, phosphates, and sulfides (OME, 1993). Under anaerobic conditions in sediments, copper primarily forms sulfide complexes and becomes immobile. Under aerobic conditions in sediments, copper is mainly present in organic complexes or bound to manganese and iron oxides (OME, 1993). Toxicity of copper appears to be a function of calcium hardness and associated carbonate alkalinity.

Bioavailability and toxicity of copper to aquatic organisms is dependent on the total concentration of copper and its chemical form. Both bioavailability and toxicity are significantly reduced by increases in suspended solids, water hardness, and the presence of natural organic chelators (Eisler, 1998). Copper is not known to be appreciably bioaccumulated by fish, but some algae and bivalve mollusks do bioconcentrate or bioaccumulate copper by factors of over 1000 (USEPA, 1985). Bioconcentration factors reported for several marine invertebrate species range from 90 for the mussel (Mytilus edulis) in a 14-day study to 3,300 for the clam (Mya arenaria) in a 35-day study (Boening, 1998). BCFs in freshwater ranged from zero in the bluegill (Lepomis macrochirus) to 2,000 in algae (Boening, 1998). It is generally assumed that copper does not significantly biomagnify in food chains (Boening, 1998).

Copper is toxic to many fish and aquatic organisms. The gill is the primary organ for concentration of, and exposure to, copper in aquatic organisms. In general, early life stages are most susceptible to copper toxicity. Toxicity to aquatic life is related primarily to the dissolved cupric ion. The cupric ion (2+) is the most readily available and toxic inorganic species of copper in freshwater, seawater, and sediment interstitial waters. In solution, copper interacts with numerous inorganic and organic compounds resulting in altered bioavailability and toxicity (Eisler, 1998). Copper toxicity is dependent on water hardness, decreasing as hardness increases. Increased temperature has the effect of decreasing the toxicity of copper (Mance, 1990).

Screening values for birds and mammals were based on studies reviewed in Sample et al. (1996). A 357-day study on the effects of copper on the reproduction of mink indicated increased mortality of mink kits at oral doses of 50, 100, and 200 ppm (Aulerich et al., 1982). A chronic NOAEL of 11.7 mg/kg/day was determined from the 25 ppm dietary concentration at which no adverse reproductive effects were observed. A 10-week study on the effects of copper on the growth and mortality of day old chicks indicated reduced growth and increased mortality at a dietary concentration of 749 ppm (Mehring et al., 1960). No adverse effects were observed at a dietary concentration of 570 ppm that was converted to a daily dose of 47 mg/kg/day.

Lead

The toxicity profile for lead was taken from USEPA (2005c). Lead is cancer-causing, and adversely affects reproduction, liver and thyroid function, and disease resistance (Eisler, 1988). Lead partitions primarily to sediments, but becomes more bioavailable under low pH, hardness and organic matter content (among other factors). Organic forms of lead are more bioavailable than inorganic forms, but microorganisms in streams are capable of transforming inorganic lead into organic forms. Soluble lead is toxic to all aquatic plant phyla. Lead adversely affects algae, invertebrates, and fish. Fish exposed to high levels of lead exhibit a wide-range of effects including muscular and neurological degeneration and destruction, growth inhibition, mortality, reproductive problems, and paralysis (Eisler, 1988; USEPA, 1976). Lead bioaccumulates in algae, macrophytes and benthic organisms, but the inorganic forms of lead do not biomagnify. Lead adversely affects invertebrate reproduction and algal growth.

Birds and mammals suffer effects from lead poisoning such as damage to the nervous system, kidneys, liver, sterility, growth inhibition, developmental retardation, and detrimental effects in blood (Eisler, 1988b; Amdur et al., 1991). Lead poisoning in higher organisms has been associated with lead shot and organolead compounds, but not with food chain exposure to inorganic lead (other than lead shot, sinkers or paint) (Eisler, 1988b). There are complex interactions with other contaminants and diet. Lead poisoning in higher organisms primarily affects hematologic and neurologic processes.

Screening values for birds and mammals were based on studies reviewed in Sample et al. (1996). A study on three generations of rats fed lead acetate indicated a chronic NOAEL of 8 mg/kg/day (Azar et al., 1973). Rats fed this dose level were not observed to exhibit any adverse reproductive effects. Rats fed 80 mg/kg/day were observed to have reduced offspring weights and kidney damage in the young. A 7-month study on the toxicological effects of lead ingestion in American kestrels found that an oral dose of 3.85 mg/kg/day did

not cause any adverse reproductive effects. A 12-week study with Japanese quail found that oral exposures to lead acetate in the diet did not have any adverse reproductive effects at doses of 1.13 mg/kg/day although adverse effects were observed at a dose of 11.3 mg/kg/day.

Zinc

Zinc, like many other metals, is essential in cell growth and enzymatic formation. Ceriodaphnia, a genus of aquatic invertebrates, are the most sensitive of 35 genera tested, but some aquatic plants are three times as sensitive to zinc. In many types of aquatic plants and animals, growth, survival, and reproduction can all be adversely affected by elevated zinc levels (USEPA, 2005c; Eisler, 1993). Zinc in aquatic systems tends to be partitioned into sediment and less frequently dissolved as hydrated zinc ions and organic and inorganic complexes (USEPA, 2005c; MacDonald, 1993). Zinc toxicity can result in destruction of gill epithelium and tissue hypoxia in fish. Zinc is not known to magnify in food chains because the body regulates it and excess zinc is eliminated.

Screening values for birds and mammals were based on studies reviewed in ATSDR (1994) and Sample et al. (1996). Mink exposed to zinc in the diet for 25 weeks did not exhibit any adverse reproductive effects at a daily dose of 20.8 mg/kg/day. Reproduction in chickens exposed to zinc in the diet for 44 weeks was not adversely affected at a daily dose of 14.5 mg/kg/day but was adversely affected at 131 mg/kg/day.

A.2 Human Health

See attachments

Toxicity Profiles

Toxicity Summary for ANTIMONY

NOTE: Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the current toxicity values.

DECEMBER 1992

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

Antimony (Sb) is a naturally occurring metal that is used in various manufacturing processes. It exists in valence states of 3 and 5 (Budavari, 1989; ATSDR, 1990). Antimony is a common urban air pollutant (Beliles, 1979). Exposure to antimony may be via inhalation, oral and dermal routes (ATSDR, 1990).

Antimony is sparingly absorbed following ingestion or inhalation (Felicetti et al., 1974a; Gerber et al., 1982; ATSDR, 1990). Both gastrointestinal and pulmonary absorption are a function of compound solubility. Antimony is transported in the blood, its distribution varying among species and dependent on its valence state (Felicetti et al., 1974b). Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups (ATSDR, 1990). Excretion of antimony is primarily via the urine and feces, and is also dependent upon valence state (Cooper et al., 1968; Ludersdorf et al., 1987; ATSDR, 1990).

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting, diarrhea), respiratory difficulties, and death at extremely high doses (Bradley and Frederick, 1941; Beliles, 1979; ATSDR, 1990). Subchronic and chronic oral exposure may affect hematologic parameters (ATSDR, 1990). Long-term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals (Schroeder et al., 1970). Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses (Marmo et al., 1987; Rossi et al., 1987).

Acute inhalation exposure of humans may cause gastrointestinal disorders (probably due to ingestion of airborne antimony) (ATSDR, 1990). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death (Price et al., 1979). Long-term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality (Renes, 1953; Breiger et al., 1954). Antimony levels for these occupational exposure evaluations ranged from 2.2 to 11.98 mg Sb/m^3. Based on limited data, occupational exposure of women to metallic antimony and several antimonials has reportedly caused alterations in the menstrual cycle and an increased incidence of spontaneous abortions (Belyaeva, 1967). Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide (Belyaeva, 1967).

No data were available indicating that dermal exposure of humans to antimony or its compounds results in adverse effects. However dermal application of high doses of antimony oxide (1,584 mg Sb/kg) resulted in the death of rabbits within one day (IBTL, 1972). Eye irritation due to exposure to

stibine gas and several antimony oxides has been reported for humans (Stevenson, 1965; Potkonjak and Pavlovich, 1983).

The U. S. EPA (U.S. EPA, 1991, 1992) has calculated subchronic and chronic oral reference doses (RfDs) of 4E-4 mg/kg/day based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in the drinking water (5 ppm equivalent to 0.35 mg Sb/kg/day). An uncertainty factor of 1,000 was applied: 10 for extrapolation from a lowest-observed-adverse-effect-level (LOAEL) to a no-observed-adverse-effect-level (NOAEL), 10 for extrapolation from animal data, and 10 for protection of sensitive populations.

The primary target organ for acute oral exposure to antimony appears to be the gastrointestinal tract (irritation, diarrhea, vomiting) and targets for long-term exposure are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR, 1990). Inhalation exposure to antimony affects the respiratory tract (pneumoconiosis, restrictive airway disorders), with secondary targets being the cardiovascular system (altered blood pressure and electrocardiograms) and kidneys (histological changes) (Renes, 1953; Breiger et al., 1954). Only limited evidence exists for reproductive disorders due to antimony exposure (Belyaeva, 1967).

Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors (Wong et al., 1979; Watt, 1980; Groth et al., 1986; Bio/dynamics, 1989), the U.S. EPA has not evaluated antimony or antimonials for carcinogenicity and a Weight-of-Evidence classification is currently unavailable.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

ANTIMONY
CAS # 7440-36-0

Agency for Toxic Substances and Disease Registry ToxFAQs **September 1995**

This fact sheet answers the most frequently asked health questions (FAQs) about antimony. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to antimony occurs in the workplace or from skin contact with soil at hazardous waste sites. Breathing high levels of antimony for a long time can irritate the eyes and lungs, and can cause problems with the lungs, heart, and stomach. This chemical has been found in at least 403 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is antimony?

(Pronounced ăn′tə-mō′nē)

Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then mixed with other metals to form antimony alloys or combined with oxygen to form antimony oxide.

Little antimony is currently mined in the United States. It is brought into this country from other countries for processing. However, there are companies in the United States that produce antimony as a by-product of smelting lead and other metals.

Antimony isn't used alone because it breaks easily, but when mixed into alloys, it is used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, and pewter. Antimony oxide is added to textiles and plastics to prevent them from catching fire. It is also used in paints, ceramics, and fireworks, and as enamels for plastics, metal, and glass.

What happens to antimony when it enters the environment?

- Antimony is released to the environment from natural sources and from industry.
- In the air, antimony is attached to very small particles that may stay in the air for many days.
- Most antimony ends up in soil, where it attaches strongly to particles that contain iron, manganese, or aluminum.
- Antimony is found at low levels in some rivers, lakes, and streams.

How might I be exposed to antimony?

- Because antimony is found naturally in the environment, the general population is exposed to low levels of it every day, primarily in food, drinking water, and air.
- It may be found in air near industries that process or release it, such as smelters, coal-fired plants, and refuse incinerators.
- In polluted areas containing high levels of antimony, it may be found in the air, water, and soil.
- Workers in industries that process it or use antimony ore may be exposed to higher levels.

How can antimony affect my health?

Exposure to antimony at high levels can result in a variety of adverse health effects.

Breathing high levels for a long time can irritate your eyes and lungs and can cause heart and lung problems, stomach pain, diarrhea, vomiting, and stomach ulcers.

In short-term studies, animals that breathed very high levels of antimony died. Animals that breathed high levels

had lung, heart, liver, and kidney damage. In long-term studies, animals that breathed very low levels of antimony had eye irritation, hair loss, lung damage, and heart problems. Problems with fertility were also noted. In animal studies, problems with fertility have been seen when rats breathed very high levels of antimony for a few months.

Ingesting large doses of antimony can cause vomiting. We don't know what other effects may be caused by ingesting it. Long-term animal studies have reported liver damage and blood changes when animals ingested antimony. Antimony can irritate the skin if it is left on it.

Antimony can have beneficial effects when used for medical reasons. It has been used as a medicine to treat people infected with parasites.

How likely is antimony to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified antimony as to its human carcinogenicity.

Lung cancer has been observed in some studies of rats that breathed high levels of antimony. No human studies are available. We don't know whether antimony will cause cancer in people.

Is there a medical test to show whether I've been exposed to antimony?

Tests are available to measure antimony levels in the body. Antimony can be measured in the urine, feces, and blood for several days after exposure. However, these tests cannot tell you how much antimony you have been exposed to or whether you will experience any health effects. Some tests are not usually performed in most doctors' offices and may require special equipment to conduct them.

Has the federal government made recommendations to protect human health?

The EPA allows 0.006 parts of antimony per million parts of drinking water (0.006 ppm). The EPA requires that discharges or spills into the environment of 5,000 pounds or more of antimony be reported.

The Occupational Safety and Health Administration **(**OSHA**)** has set an occupational exposure limit of 0.5 milligrams of antimony per cubic meter of air (0.5 mg/m^3) for an 8-hour workday, 40-hour workweek.

The American Conference of Governmental Industrial Hygienists (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) currently recommend the same guidelines for the workplace as OSHA.

Glossary

Carcinogenicity: Ability to cause cancer.
CAS: Chemical Abstracts Service.
Ingestion: Taking food or drink into your body.
Long-term: Lasting one year or more.
Milligram (mg): One thousandth of a gram.
Parasite: An organism living in or on another organism.
ppm: Parts per million.
Short-term: Lasting 14 days or less.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for antimony. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Toxicity Profiles

Toxicity Summary for COPPER

NOTE: Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the current toxicity values.

DECEMBER 1992

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Prepared for: Oak Ridge Reservation Environmental Restoration Program.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH, 1986). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR, 1990; U.S. EPA, 1987).

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (U.S. AF, 1990). Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu/L (Mueller-Hoecker et al., 1988; Spitalny et al., 1984). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. The clinical manifestations of Wilson's disease include cirrhosis of the liver, hemolytic anemia, neurologic abnormalities, and corneal opacities (Goyer, 1991; ATSDR, 1990; U.S. EPA, 1987). In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of >=100 mg/kg/day; and hematological effects at doses of 40 mg/kg/day (U.S. EPA, 1986; Haywood, 1985; 1980; Rana and Kumar, 1978; Gopinath et al., 1974; Kline et al., 1971).

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m^3 may cause metal fume fever with symptoms such as cough, chills and muscle ache (U.S. AF, 1990). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly (Suciu et al., 1981). Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR, 1990).

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals (Lecyk, 1980; Ferm and Hanlon, 1974). Evidence also indicates that copper compounds are spermicidal (ATSDR, 1990; Battersby et al., 1982).

A Reference Dose (RfD) for elemental copper is not available (U.S. EPA, 1992). However, EPA established an action level of 1300 ug/L for drinking water (56 FR 26460, June 7, 1991). Data were insufficient to derive a Reference concentration (RfC) for copper.

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper. Therefore, U.S. EPA (1991a) has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

COPPER
CAS # 7440-50-8

Division of Toxicology ToxFAQs™ **September 2004**

This fact sheet answers the most frequently asked health questions (FAQs) about copper. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Copper is a metal that occurs naturally in the environment, and also in plants and animals. Low levels of copper are essential for maintaining good health. High levels can cause harmful effects such as irritation of the nose, mouth and eyes, vomiting, diarrhea, stomach cramps, nausea, and even death. Copper has been found in at least 906 of the 1,647 National Priority Sites identified by the Environmental Protection Agency (EPA).

What is copper?

Copper is a metal that occurs naturally throughout the environment, in rocks, soil, water, and air. Copper is an essential element in plants and animals (including humans), which means it is necessary for us to live. Therefore, plants and animals must absorb some copper from eating, drinking, and breathing.

Copper is used to make many different kinds of products like wire, plumbing pipes, and sheet metal. U.S. pennies made before 1982 are made of copper, while those made after 1982 are only coated with copper. Copper is also combined with other metals to make brass and bronze pipes and faucets.

Copper compounds are commonly used in agriculture to treat plant diseases like mildew, for water treatment and, as preservatives for wood, leather, and fabrics.

What happens to copper when it enters the environment?

❏ Copper is released into the environment by mining, farming, and manufacturing operations and through waste water releases into rivers and lakes. Copper is also released from natural sources, like volcanoes, windblown dusts, decaying vegetation, and forest fires.

❏ Copper released into the environment usually attaches to particles made of organic matter, clay, soil, or sand.

❏ Copper does not break down in the environment. Copper compounds can break down and release free copper into the air, water, and foods.

How might I be exposed to copper?

❏ You may be exposed to copper from breathing air, drinking water, eating foods, or having skin contact with copper, particulates attached to copper, or copper-containing compounds.

❏ Drinking water may have high levels of copper if your house has copper pipes and acidic water.

❏ Lakes and rivers that have been treated with copper compounds to control algae, or that receive cooling water from power plants, can have high levels of copper. Soils can also contain high levels of copper, especially if they are near copper smelting plants.

❏ You may be exposed to copper by ingesting copper-containing fungicides, or if you live near a copper mine or where copper is processed into bronze or brass.

❏ You may be exposed to copper if you work in copper mines or if you grind metals containing copper.

How can copper affect my health?

Everyone must absorb small amounts of copper every day because copper is essential for good health. High levels of copper can be harmful. Breathing high levels of copper can cause irritation of your nose and throat. Ingesting high levels of copper can cause nausea, vomiting, and diarrhea. Very-high doses of copper can cause damage to your liver and kidneys, and can even cause death.

How likely is copper to cause cancer?

We do not know whether copper can cause cancer in humans. The EPA has determined that copper is not classifiable as to human carcinogenicity.

How can copper affect children?

Exposure to high levels of copper will result in the same type of effects in children and adults. We do not know if these effects would occur at the same dose level in children and adults. Studies in animals suggest that the young children may have more severe effects than adults, but we don't know if this would also be true in humans. There is a very small percentage of infants and children who are unusually sensitive to copper.

We do not know if copper can cause birth defects or other developmental effects in humans. Studies in animals suggest that high levels of copper may cause a decrease in fetal growth.

How can families reduce the risk of exposure to copper?

The most likely place to be exposed to copper is through drinking water, especially if your water is corrosive and you have copper pipes in your house. The best way to lower the level of copper in your drinking water is to let the water run for at least 15 seconds first thing in the morning before drinking or using it. This reduces the levels of copper in tap water dramatically.

If you work with copper, wear the necessary protective clothing and equipment, and always follow safety procedures. Shower and change your clothes before going home each day.

Is there a medical test to show whether I've been exposed to copper?

Copper is found throughout the body; in hair, nails, blood, urine, and other tissues. High levels of copper in these samples can show that you have been exposed to higher-than normal levels of copper. These tests cannot tell whether you will experience harmful effects. Tests to measure copper levels in the body are not usually available at a doctor's office because they require special equipment, but the doctor can send samples to a specialty laboratory.

Has the federal government made recommendations to protect human health?

The EPA requires that levels of copper in drinking water be less than 1.3 mg of copper per one liter of drinking water (1.3 mg/L).

The U.S. Department of Agriculture has set the recommended daily allowance for copper at 900 micrograms of copper per day (µg/day) for people older than eight years old.

The Occupational Safety and Health Administration (OSHA) requires that levels of copper in the air in workplaces not exceed 0.1 mg of copper fumes per cubic meter of air ($0.1\ mg/m^3$) and $1.0\ mg/m^3$ for copper dusts.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile for Copper. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Toxicity Profiles

Toxicity Summary for LEAD

NOTE: Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the current toxicity values.

December 1994

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM.

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Lead occurs naturally as a sulfide in galena. It is a soft, bluish-white, silvery gray, malleable metal with a melting point of 327.5C. Elemental lead reacts with hot boiling acids and is attacked by pure water. The solubility of lead salts in water varies from insoluble to soluble depending on the type of salt (IARC, 1980; Goyer, 1988; Budavari et al., 1989).

Lead is a natural element that is persistent in water and soil. Most of the lead in environmental media is of anthropogenic sources. The mean concentration is 3.9 ug/L in surface water and 0.005 ug/L in sea water. River sediments contain about 20,000 ug/g and coastal sediments about 100,000 ug/g. Soil content varies with the location, ranging up to 30 ug/g in rural areas, 3000 ug/g in urban areas, and 20,000 ug/g near point sources. Human exposure occurs primarily through diet, air, drinking water, and ingestion of dirt and paint chips (EPA, 1989; ATSDR, 1993).

The efficiency of lead absorption depends on the route of exposure, age, and nutritional status. Adult humans absorb about 10-15% of ingested lead, whereas children may absorb up to 50%, depending on whether lead is in the diet, dirt, or paint chips. More than 90% of lead particles deposited in the respiratory tract are absorbed into systemic circulation. Inorganic lead is not efficiently absorbed through the skin; consequently, this route does not contribute considerably to the total body lead burden (EPA, 1986a).

Lead absorbed into the body is distributed to three major compartments: blood, soft tissue, and bone. The largest compartment is the bone, which contains about 95% of the total body lead burden in adults and about 73% in children. The half-life of bone lead is more than 20 years. The concentration of blood lead changes rapidly with exposure, and its half-life of only 25-28 days is considerably shorter than that of bone lead. Blood lead is in equilibrium with lead in bone and soft tissue. The soft tissues that take up lead are liver, kidneys, brain, and muscle. Lead is not metabolized in the body, but it may be conjugated with glutathione and excreted primarily in the urine (EPA, 1986a,c; ATSDR, 1993). Exposure to lead is evidenced by elevated blood lead levels.

The systemic toxic effects of lead in humans have been well-documented by the EPA (EPA, 1986a-e, 1989a, 1990) and ATSDR (1993), who extensively reviewed and evaluated data reported in the literature up to 1991. The evidence shows that lead is a multitargeted toxicant, causing effects in the gastrointestinal tract, hematopoietic system, cardiovascular system, central and peripheral nervous systems, kidneys, immune system, and reproductive system. Overt symptoms of subencephalopathic central nervous system (CNS) effects and peripheral nerve damage occur at blood lead levels of 40-60 ug/dL, and nonovert symptoms, such as peripheral nerve dysfunction, occur at levels of 30-50 ug/dL in adults; no clear threshold is evident. Cognitive and neuropsychological deficits are not usually the focus of studies in adults, but there is some evidence of neuropsychological impairment

(Ehle and McKee, 1990) and cognitive deficits in lead workers with blood levels of 41-80 ug/dL (Stollery et al., 1993).

Although similar effects occur in adults and children, children are more sensitive to lead exposure than are adults. Irreversible brain damage occurs at blood lead levels greater than or equal to 100 ug/dL in adults and at 80-100 ug/dL in children; death can occur at the same blood levels in children. Children who survive these high levels of exposure suffer permanent severe mental retardation.

As discussed previously, neuropsychological impairment and cognitive (IQ) deficits are sensitive indicators of lead exposure; both neuropsychological impairment and IQ deficits have been the subject of cross-sectional and longitudinal studies in children. One of the early studies reported IQ score deficits of four points at blood lead levels of 30-50 ug/dL and one to two points at levels of 15-30 ug/dL among 75 black children of low socioeconomic status (Schroeder and Hawk, 1986).

Very detailed longitudinal studies have been conducted on children (starting at the time of birth) living in Port Pirie, Australia (Vimpani et al., 1985, 1989; McMichael et al., 1988; Wigg et al., 1988; Baghurst et al., 1992a,b), Cincinnati, Ohio (Dietrich et al., 1986, 1991, 1992, 1993), and Boston, Massachusetts (Bellinger et al., 1984, 1987, 1990, 1992; Stiles and Bellinger 1993). Various measures of cognitive performance have been assessed in these children. Studies of the Port Pirie children up to 7 years of age revealed IQ deficits in 2-year-old children of 1.6 points for each 10-ug/dL increase in blood lead, deficits of 7.2 points in 4-year-old children, and deficits of 4.4 to 5.3 points in 7-year-old children as blood lead increased from 10-30 ug/dL. No significant neurobehavioral deficits were noted for children, 5 years or younger, who lived in the Cincinnati, Ohio, area. In 6.5-year-old children, performance IQ was reduced by 7 points in children whose lifetime blood level exceeded 20 ug/dL.

Children living in the Boston, Massachusetts, area have been studied up to the age of 10 years. Cognitive performance scores were negatively correlated with blood lead in the younger children in the high lead group (greater than or equal to 10 ug/dL), and improvements were noted in some children at 57 months as their blood lead levels became lower. However, measures of IQ and academic performance in 10-year-old children showed a 5.8-point deficit in IQ and an 8.9-point deficit in academic performance as blood lead increased by 10 ug/dL within the range of 1-25 ug/dL. Because of the large database on subclinical neurotoxic effects of lead in children, only a few of the studies have been included. However, EPA (EPA, 1986a, 1990) concluded that there is no clear threshold for neurotoxic effects of lead in children.

In adults, the cardiovascular system is a very sensitive target for lead. Hypertension (elevated blood pressure) is linked to lead exposure in occupationally exposed subjects and in the general population. Three large population-based studies have been conducted to study the relationship between blood lead levels and high blood pressure. The British Regional Heart Study (BRHS) (Popcock et al., 1984), the NHANES II study (Harlan et al., 1985; Pirkle et al., 1985; Landis and Flegal, 1988; Schwartz, 1990; EPA, 1990), and Welsh Heart Programme (Ellwood et al., 1988a,b) comprise the major studies for the general population. The BRHS study showed that systolic pressure greater than 160 mm Hg and diastolic pressure greater than 100 mm Hg were associated with blood lead levels greater than 37 ug/dL (Popcock et al., 1984). An analysis of 9933 subjects in the NHANES study showed positive correlations between blood pressure and blood lead among 12-74-year-old males but not females (Harlan et al., 1985; Landis and Flegal et al., 1988), 40-59-year-old white males with blood levels ranging from 7-34 ug/dL (Pirkle et al., 1985), and males and females greater than 20 years old (Schwartz, 1991). In addition, left ventricular hypertrophy was also positively associated with blood lead (Schwartz, 1991). The Welsh study did not show an association among men and women with blood lead of 12.4 and 9.6 ug/dL, respectively (Ellwood et al., 1988a,b). Other smaller studies showed both positive and negative results. The EPA (EPA, 1990) concluded that increased blood pressure is positively correlated with blood lead levels in middle-aged men, possibly at concentrations as low as 7 ug/dL. In addition, the EPA estimated that systolic pressure is increased by 1.5-3.0 mm Hg in males and 1.0-2.0 mm Hg in females for every doubling of blood lead concentration.

The hematopoietic system is a target for lead as evidenced by frank anemia occurring at blood lead levels of 80 ug/dL in adults and 70 ug/dL in children. The anemia is due primarily to reduced heme synthesis, which is observed in adults having blood levels of 50 ug/dL and in children having blood levels of 40 ug/dL. Reduced heme synthesis is caused by inhibition of key enzymes involved in the synthesis of heme. Inhibition of erythrocyte -aminolevulinic acid dehydrase (ALAD) activity (catalyzes formation of porphobilinogen from -aminolevulinic acid) has been detected in adults and children having blood levels of less than 10 ug/dL. ALAD activity is the most sensitive measure of lead exposure, but erythrocyte zinc protoporphyrin is the most reliable indicator of lead exposure because it is a measure of the toxicologically active fraction of bone lead. The activity of another erythrocyte enzyme, pyrimidine-5-nucleotidase, is also inhibited by lead exposure. Inhibition has been observed at levels below 5 ug/dL; no clear threshold is evident.

Other organs or systems affected by exposure to lead are the kidneys, immune system, reproductive system, gastrointestinal tract, and liver. These effects usually occur at high blood levels, or the blood levels at which they occur have not been sufficiently documented.

The EPA has not developed an RfD for lead because it appears that lead is a nonthreshold toxicant, and it is not appropriate to develop RfDs for these types of toxicants. Instead the EPA has developed the Integrated Exposure Uptake Biokenetic Model to estimate the percentage of the population of children up to 6 years of age with blood lead levels above a critical value, 10 ug/dL. The model determines the contribution of lead intake from multimedia sources (diet, soil and dirt, air, and drinking water) on the concentration of lead in the blood. Site-specific concentrations of lead in various media are used when available; otherwise default values are assumed. The EPA has established a screening level of 400 ppm (ug/g) for lead in soil (EPA, 1994a).

Inorganic lead and lead compounds have been evaluated for carcinogenicity by the EPA (EPA, 1989, 1993). The data from human studies are inadequate for evaluating the potential carcinogenicity of lead. Data from animal studies, however, are sufficient based on numerous studies showing that lead induces renal tumors in experimental animals. A few studies have shown evidence for induction of tumors at other sites (cerebral gliomas; testicular, adrenal, prostate, pituitary, and thyroid tumors). A slope factor was not derived for inorganic lead or lead compounds.

ATSDR
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LEAD
CAS # 7439-92-1

This fact sheet answers the most frequently asked health questions (FAQs) about lead. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to lead can happen from breathing workplace air or dust, eating contaminated foods, or drinking contaminated water. Children can be exposed from eating lead-based paint chips or playing in contaminated soil. Lead can damage the nervous system, kidneys, and reproductive system. Lead has been found in at least 1,026 of 1,467 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is lead?

(Pronounced lĕd)

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. Lead can be found in all parts of our environment. Much of it comes from human activities including burning fossil fuels, mining, and manufacturing.

Lead has many different uses. It is used in the production of batteries, ammunition, metal products (solder and pipes), and devices to shield X-rays.

Because of health concerns, lead from gasoline, paints and ceramic products, caulking, and pipe solder has been dramatically reduced in recent years.

What happens to lead when it enters the environment?

- Lead itself does not break down, but lead compounds are changed by sunlight, air, and water.
- When lead is released to the air, it may travel long distances before settling to the ground.
- Once lead falls onto soil, it usually sticks to soil particles.
- Movement of lead from soil into groundwater will depend on the type of lead compound and the characteristics of the soil.
- Much of the lead in inner-city soils comes from old houses painted with lead-based paint.

How might I be exposed to lead?

- Eating food or drinking water that contains lead.
- Spending time in areas where lead-based paints have been used and are deteriorating.
- Working in a job where lead is used.
- Using health-care products or folk remedies that contain lead.
- Engaging in certain hobbies in which lead is used (for example, stained glass).

How can lead affect my health?

Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The effects are the same whether it is breathed or swallowed.

At high levels, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain.

How likely is lead to cause cancer?

The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably

be anticipated to be carcinogens based on studies in animals. There is inadequate evidence to clearly determine lead's carcinogenicity in people.

How can lead affect children?

Small children can be exposed by eating lead-based paint chips, chewing on objects painted with lead-based paint, or swallowing house dust or soil that contains lead.

Children are more vulnerable to lead poisoning than adults. A child who swallows large amounts of lead may develop blood anemia, severe stomachache, muscle weakness, and brain damage. A large amount of lead might get into a child's body if the child ate small pieces of old paint that contained large amounts of lead. If a child swallows smaller amounts of lead, much less severe effects on blood and brain function may occur. Even at much lower levels of exposure, lead can affect a child's mental and physical growth.

Exposure to lead is more dangerous for young and unborn children. Unborn children can be exposed to lead through their mothers. Harmful effects include premature births, smaller babies, decreased mental ability in the infant, learning difficulties, and reduced growth in young children. These effects are more common if the mother or baby was exposed to high levels of lead.

How can families reduce the risk of exposure to lead?

Avoid exposure to sources of lead. Do not allow children to chew or mouth painted surfaces that may have been painted with lead-based paint (homes built before 1978). Run your water for 15 to 30 seconds before drinking or cooking with it. This will get rid of lead that may have leached out of pipes. Some types of paints and pigments that are used as make-up or hair coloring contain lead. Keep these kinds of products away from children. Wash children's hands and faces often to remove lead dusts and soil, and regularly clean the house of dust and tracked in soil.

Is there a medical test to show whether I've been exposed to lead?

A blood test is available to measure the amount of lead in your blood and to estimate the amount of your exposure to lead. Blood tests are commonly used to screen children for lead poisoning. Lead in teeth and bones can be measured with X-rays, but this test is not as readily available. Medical treatment may be necessary in children if the lead concentration in blood is higher than 45 micrograms per deciliter (45 µg/dL).

Has the federal government made recommendations to protect human health?

The Centers for Disease Control and Prevention (CDC) recommends that children ages 1 and 2 be screened for lead poisoning. Children who are 3 to 6 years old should be tested for lead if they have never been tested for lead before and if they receive services from public assistance programs; if they live in or regularly visit a building built before 1950; if they live in or visit a home built before 1978 that is being remodeled; or if they have a brother, sister, or playmate who has had lead poisoning. CDC considers children to have an elevated level of lead if the amount in the blood is 10 µg/dL.

The EPA requires lead in air not to exceed 1.5 micrograms per cubic meter (1.5 $\mu g/m^3$) averaged over 3 months. EPA limits lead in drinking water to 15 µg per liter.

The Occupational Health and Safety Administration (OSHA) develops regulations for workers exposed to lead. The Clean Air Act Amendments of 1990 banned the sale of leaded gasoline. The Federal Hazardous Substance Act bans children's products that contain hazardous amounts of lead.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. Toxicological profile for lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Toxicity Profiles

Toxicity Summary for ZINC AND ZINC COMPOUNDS

NOTE: Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the current toxicity values.

April 1992

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400

Zinc is used primarily in galvanized metals and metal alloys, but zinc compounds also have wide commercial applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. They are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd, 1984; ATSDR, 1989). In addition, zinc phosphide is used as a rodenticide.

Zinc is an essential element with recommended daily allowances ranging from 5 mg for infants to 15 mg for adult males (NRC, 1989).

Gastrointestinal absorption of zinc is variable (20-80%) and depends on the chemical compound as well as on zinc levels in the body and dietary concentrations of other nutrients (U.S. EPA, 1984). In individuals with normal zinc levels in the body, gastrointestinal absorption is 20-30% (ATSDR, 1989). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO_2 exchange (Stokinger, 1981). Homeostatic mechanisms involving metallothionein in the mucosal cells of the gastrointestinal tract regulate zinc absorption and excretion (ATSDR, 1989).

In humans, acutely toxic oral doses of zinc cause nausea, vomiting, diarrhea, and abdominal cramps and in some cases gastric bleeding (Elinder, 1986; Moore, 1978; ATSDR, 1989). Ingestion of zinc chloride can cause burning in the mouth and throat, vomiting, pharyngitis, esophagitis, hypocalcemia, and elevated amylase activity indicative of pancreatitis (Chobanian, 1981). Zinc phosphide, which releases phosphine gas under acidic conditions in the stomach, can cause vomiting, anorexia, abdominal pain, lethargy, hypotension, cardiac arrhythmias, circulatory collapse, pulmonary edema, seizures, renal damage, leukopenia, and coma and death in days to weeks (Mack, 1989). The estimated fatal dose is 40 mg/kg. Animals dosed orally with zinc compounds develop pancreatitis, gastrointestinal and hepatic lesions, and diffuse nephrosis.

Gastrointestinal upset has also been reported in individuals taking daily dietary zinc supplements for up to 6 weeks (Samman and Roberts, 1987). There is also limited evidence that the human immune system may be impaired by subchronic exposures (Chandra, 1984). In animals, gastrointestinal and

hepatic lesions, (Allen et al., 1983; Brink et al., 1959); pancreatic lesions (Maita et al., 1981; Drinker et al., 1927a); anemia (ATSDR, 1989; Fox and Jacobs, 1986; Maita et al., 1981); and diffuse nephrosis (Maita et al., 1981; Allen et al., 1983) have been observed following subchronic oral exposures.

Chronic oral exposures to zinc have resulted in hypochromic microcytic anemia associated with hypoceruloplasminemia, hypocupremia, and neutropenia in some individuals (Prasad et al., 1978; Porter et al., 1977). Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al., 1977; Drinker et al., 1927a; Walters and Roe, 1965; Sutton and Nelson, 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al., 1969; Schlicker and Cox 1967, 1968; Sutton and Nelson, 1937).

The reference dose for chronic oral exposure to zinc is under review by EPA; the currently accepted RfD for both subchronic and chronic exposures is 0.2 mg/kg/day based on clinical data demonstrating zinc-induced copper deficiency and anemia in patients taking zinc sulfate for the treatment of sickle cell anemia (U.S. EPA, 1992). The chronic oral RfD for zinc phosphide is 0.0003 mg/kg/day (U.S. EPA, 1991a), and the subchronic RfD is 0.003 mg/kg/day (U.S. EPA, 1992).

Under occupational exposure conditions, inhalation of zinc compounds (mainly zinc oxide fumes) can result in a condition identified as "metal fume fever", which is characterized by nasal passage irritation, cough, rales, headache, altered taste, fever, weakness, hyperpnea, sweating, pains in the legs and chest, leukocytosis, reduced lung volume, and decreased diffusing capacity of carbon monoxide (ATSDR, 1989; Bertholf, 1988). Inhalation of zinc chloride can result in nose and throat irritation, dyspnea, cough, chest pain, headache, fever, nausea and vomiting, and respiratory disorders such as pneumonitis and pulmonary fibrosis (ITII, 1988; ATSDR, 1989; Nemery, 1990). Pulmonary inflammation and changes in lung function have also been observed in inhalation studies on animals (Amur et al., 1982; Lam et al., 1985; Drinker and Drinker, 1928).

Although "metal fume fever" occurs in occupationally exposed workers, it is primarily an acute and reversible effect that is unlikely to occur under chronic exposure conditions when zinc air concentrations are less than 8-12 mg/m^3 (ATSDR, 1989). Gastrointestinal distress, as well as enzyme changes indicative of liver dysfunction, have also been reported in workers occupationally exposed to zinc (NRC, 1979; Stokinger, 1981; U.S. EPA, 1991a; Guja, 1973; Badawy et al., 1987a); however, it is unclear as to what extent these effects might have been caused by pulmonary clearance, and subsequent gastrointestinal absorption. Consequently, there are no clearly defined toxic effects that can be identified as resulting specifically from pulmonary absorption following chronic low level inhalation exposures. Animal data for chronic inhalation exposures are not available.

An inhalation reference concentration has not been derived for zinc or zinc compounds (U.S. EPA, 1992).

No case studies or epidemiologic evidence has been presented to suggest that zinc is carcinogenic in humans by the oral or inhalation route (U.S. EPA, 1991a). In animal studies, zinc sulfate in drinking water or zinc oleate in the diet of mice for a period of one year did not result in a statistically significant increase in hepatomas, malignant lymphomas, or lung adenomas (Walters and Roe, 1965); however, in a 3-year, 5-generation study on tumor-resistant and tumor-susceptible strains of mice, exposure to zinc in drinking water resulted in increased frequencies of tumors from the F_0 to the F_4 generation in the tumor-resistant strain (from 0.8 to 25.7%, vs. 0.0004% in the controls), and higher tumor frequencies in two tumor-susceptible strains (43.4% and 32.4% vs. 15% in the controls) (Halme, 1961).

Zinc is placed in weight-of-evidence Group D, not classifiable as to human carcinogenicity due to inadequate evidence in humans and animals (U.S. EPA, 1991a).

ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

ZINC
CAS # 7440-66-6

Agency for Toxic Substances and Disease Registry ToxFAQs **September 1995**

This fact sheet answers the most frequently asked health questions (FAQs) about zinc. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to high levels of zinc occurs mostly from eating food, drinking water, or breathing workplace air that is contaminated. Exposure to large amounts of zinc can be harmful. However, zinc is an essential element for our bodies, so too little zinc can also be harmful. This chemical has been found in at least 801 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is zinc?

(Pronounced zĭngk)

Zinc is one of the most common elements in the earth's crust. It's found in air, soil, and water, and is present in all foods. Pure zinc is a bluish-white shiny metal.

Zinc has many commercial uses as coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass and bronze. A zinc and copper alloy is used to make pennies in the United States.

Zinc combines with other elements to form zinc compounds. Common zinc compounds found at hazardous waste sites include zinc chloride, zinc oxide, zinc sulfate, and zinc sulfide. Zinc compounds are widely used in industry to make paint, rubber, dye, wood preservatives, and ointments.

What happens to zinc when it enters the environment?

- Some is released into the environment by natural processes, but most comes from activities of people like mining, steel production, coal burning, and burning of waste.
- It attaches to soil, sediments, and dust particles in the air.
- Rain and snow remove zinc dust particles from the air.
- Zinc compounds can move into the groundwater and into lakes, streams, and rivers.
- Most of the zinc in soil stays bound to soil particles.
- It builds up in fish and other organisms, but it doesn't build up in plants.

How might I be exposed to zinc?

- Ingesting small amounts present in your food and water.
- Drinking contaminated water near manufacturing or waste sites.
- Drinking contaminated water or a beverage that has been stored in metal containers or flows through pipes that have been coated with zinc to resist rust.
- Eating too many dietary supplements that contain zinc.
- Breathing zinc particles in the air at manufacturing sites.

How can zinc affect my health?

Zinc is an essential element in our diet. **Too** little zinc can cause health problems, but too much zinc is also harmful.

The recommended dietary allowance (RDA) for zinc is 15 milligrams a day for men (15 mg/day); 12 mg/day for women; 10 mg/day for children; and 5 mg/day for infants. Not enough zinc in your diet can result in a loss of appetite, a decreased sense of taste and smell, slow wound healing and

skin sores, or a damaged immune system. Young men who don't get enough zinc may have poorly developed sex organs and slow growth. If a pregnant woman doesn't get enough zinc, her babies may have growth retardation.

Too much zinc, however, can also be damaging to your health. Harmful health effects generally begin at levels from 10-15 times the RDA (in the 100 to 250 mg/day range). Eating large amounts of zinc, even for a short time, can cause stomach cramps, nausea, and vomiting. Taken longer, it can cause anemia, pancreas damage, and lower levels of high density lipoprotein cholesterol (the good form of cholesterol).

Breathing large amounts of zinc (as dust or fumes) can cause a specific short-term disease called metal fume fever. This is believed to be an immune response affecting the lungs and body temperature. We do not know the long-term effects of breathing high levels of zinc.

It is not known if high levels of zinc affect human reproduction or cause birth defects. Rats that were fed large amounts of zinc became infertile or had smaller babies. Irritation was also observed on the skin of rabbits, guinea pigs, and mice when exposed to some zinc compounds. Skin irritation will probably occur in people.

How likely is zinc to cause cancer?

The Department of Health and Human Services, the International Agency for Research on Cancer, and the Environmental Protection Agency (EPA) have not classified zinc for carcinogenicity.

Is there a medical test to show whether I've been exposed to zinc?

Zinc can be measured in your blood or feces. This can tell you how much zinc you have been exposed to. Zinc can also be measured in urine, saliva, and hair. The amount of zinc in your hair tells us something about long-term exposure, but the relationship between levels in your hair and the amount that you were exposed to is not clear. These tests are not routinely performed at doctors' offices, but your doctor can take samples and send them to a testing laboratory.

Has the federal government made recommendations to protect human health?

EPA recommends that there be no more than 5 parts of zinc in 1 million parts of drinking water (5 ppm) because of taste. EPA also requires that releases of more than 1,000 (or in some cases 5,000) pounds of zinc or its compounds into the environment be reported.

The Occupational Safety and Health Administration (OSHA) has set a maximum concentration limit for zinc chloride fumes in workplace air of 1 milligram of zinc per cubic meter of air (1 mg/m^3) for an 8-hour workday over a 40-hour work week and 5 mg/m^3 for zinc oxide fumes. The National Institute for Occupational Safety and Health (NIOSH) has set the same standards for up to a 10-hour workday over a 40-hour workweek.

Glossary

Anemia: A decreased ability of the blood to transport oxygen.

Carcinogenicity: Ability to cause cancer.

Milligram (mg): One thousandth of a gram.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1994. Toxicological profile for zinc. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone:1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is http://www.atsdr.cdc.gov/toxfaq.html ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

Appendix B: Geochemical Model and Results

Surface water and Sediment Conditions

An alkaline pH (8.05) and dissolved oxygen (DO, 8 milligrams per liter [mg/L] were used for the Great Lakes, which indicates considerable oxidizing conditions. The temperature of 5 degrees Celsius (˚C) was from Kramer, 1967, who modeled the equilibrium state of the Great Lakes water chemistry. Chemical reactions will be considerably slower at this temperature than would normally occur under more standard conditions (10 to 20˚C). The reactive bed sediment minerals for the individual lakes is from Jones and Bowser (1978). There is much less chemical and particularly bed sediment mineralogy available for Chesapeake Bay. The average pH is an alkaline 8.18 indicating essentially equilibrium with respect to calcite. The average DO is a moderately oxidized 5.05 mg/L and the average modeled temperature was 14.8˚C. The average salinity of 12.86 ppt at Cedar Point was used to estimate the water chemistry by simple dilution of sea water chemistry (Langmuir, 1997) with distilled water to this salinity. The bed sediment was estimated to be the same as that of the lake sediment since the surficial bedrock lithologies surrounding Chesapeake Bay involves the same lithologies as those surrounding the lakes.

Reactive bed sediment minerals for both the lakes and Chesapeake Bay include calcite ($CaCO_3$), iron oxyhydroxide (FeOOH), hydrated manganese oxide ($Na_4Mn_{14}O_{27}{\cdot}9H_2O$) and hydrated iron orthophosphate (vivanite, $Fe_3(PO_4)_2{\cdot}8H_2O$). Of course, quartz, feldspars and clays comprise by far most of the bed sediment mineralogy. However, these minerals do not have a controlling chemical reaction on any of the metals and, in fact, will tend to significantly shield the metals and thereby further slow chemical reactions occurring from the water chemistry. Calcite largely controls the water pH of the pore water. Both the iron and manganese oxides form adsorption sites for each of the metals.

Adsorption coefficients for copper, lead and zinc are well known and acceptable for the lake and bay conditions but there are no adsorption coefficients available in the model (or any model) for antimony. This means that the only control on dissolved antimony involves the precipitation of antimony minerals (usually antimony oxide) that obviously requires oxidized conditions and high dissolved antimony concentrations. This is a serious modeling limitation for estimated dissolved antimony concentrations because antimony is really controlled by adsorption by iron and manganese oxides in nature. Therefore, the estimated antimony concentrations are significantly elevated by orders of magnitude over what they really will be in the bed sediments because antimony typically occurs at significantly lower dissolved concentrations than copper, lead or, certainly, zinc in waters with these temperature, pH and dissolved oxygen conditions.

Hydrous orthophosphate is readily available in the bed sediments and at least partially reflect microbial activity in the sediments since the orthophosphate is produced by their presence. Orthophosphate reacts with lead to form an essentially insoluble mineral (pyromorphite, $Pb_5(PO_4)_3Cl$) that along with lead carbonate (cerrusite, $PbCO_3$) and adsorption to iron and manganese oxides, significantly restrict the dissolved lead

concentration. Both copper and zinc form orthophosphate minerals also but are more likely to be precipitated as a carbonate mineral or become adsorbed to both iron and manganese oxides.

The amount of iron and manganese oxide present in the bed sediment is highly variable. Therefore, a very low (0.3 gram) to moderately low (1.0 to 2.0 grams) amount of iron oxyhydroxide in the bed sediment were modeled to illustrate the effect of the changing amount of this adsorbing material on the dissolved metals. Increasing the iron oxyhydroxide concentration tends to decrease the oxidation-reduction potential as well as the estimated dissolved copper and zinc concentrations.

The estimated dissolved metals concentrations would be orders of magnitude lower than these modeled oxidized conditions if the bed sediment oxidation-reduction potential was allowed to go to a more suitable reducing condition. Even if only moderately reducing conditions (Eh -0.1 V) were used, the metals would form sulfide minerals that release little to no dissolved metals.

Geochemical Model

The Geochemist's Workbench (Bethke, 2004) reaction path modeling program was used to model ammunition copper, lead, zinc, and antimony concentrations in Lake Superior, Lake Huron and Chesapeake Bay. Initial modeling results for Lake Michigan, Lake Erie and Lake Ontario resulted in lower concentrations than those of either Lake Superior or Lake Huron, and are therefore not evaluated further. The reaction path model incrementally adds the metals to the water and chemically reacts these metals both with the individual water chemistries and bed sediment minerals present in the lake and bay. The model produces a record of dissolved metals concentrations and metals removed by precipitation of metal-bearing minerals as well as adsorption when the metals react with both the water and the sediment mineralogy. Equilibrium conditions are established with each incremental step beginning with the initial chemical reaction (water chemistry equilibrium, reaction progress 0.0) until all the metals are added and equilibrated with the water and sediment (reaction progress 1.0 representing 100 percent). The model involves a complex suite of chemical reactions. Time is an unknown except in the context of how slowly each of the metals react with the water and sediment. Each of the metals will react at a different rate but the chemical reaction will be slow so that the ending reaction progress of 1.0 will probably be on the order of at least 10s to 100s of years.

The model is a limited portrayal of how the metals are exposed and what will happen to them. The metals will sink relatively rapidly through the water to the bottom sediments limiting their time of exposure to the lake and bay water. The metals will be exposed to the open water condition only a very limited time (the time required to sink to the bed sediments). The uppermost bed sediments are usually very soft allowing the metal to sink into the sediments. The model estimation assumes that the bed sediment pore water is the same as the lake and bay water. However, the pore water will include microbes that can significantly alter not only the water chemistry that the metals are exposed to but also the both the rate at which the metals chemically react and the minerals formed by the chemical reaction. The model assumes, at this point, that the chemical reactions are totally abiotic (no microbial activity).

The modeling estimates of dissolved metals concentrations represent a worst case condition the lakes and Chesapeake Bay. The bed sediment typically becomes reducing to highly reducing within a few centimeters depth. Reducing conditions will significantly decrease the dissolution of metals (orders of magnitude lower dissolved concentrations). Furthermore, except for the metals concentrations in the total area, there is no dilution occurring in this constant volume estimation of the dissolved metals concentrations. Effectively, modeled dissolved metals concentrations estimates assume a constant volume of water surrounding the metals (pore water). Each of the metals are also assumed to be completely exposed (i.e., no compartmentalization of bullet and slug), whereas an unknown but significant portion of the munitions will not be exposed until the more exposed portions are almost totally reacted.

Predicted Concentrations

Table B-1 lists the modeling copper, lead, zinc and antimony concentrations (mg/L) results for Lake Superior, Lake Huron and Chesapeake Bay at 100 percent of chemical reactions between the metals and both the water and bed sediment of the three. Ending pH and Eh are also listed. These results reflect the dissolved metals concentrations in essentially the pore water at the end of the chemical reactions. Most of the differences in the estimated metals concentrations result from the changing average metals concentrations in the total area.

Estimated lead concentrations have the least variability of the metals, all lead concentrations in the single digit micrograms per liter (µg/L). This is consistent with the multiple chemical reactions that control the mobility of lead. Zinc is the next most stable with variability limited to one order of magnitude in the total area (tenths to hundredths of a µg/L). Copper is more variable involving several orders of magnitude changes but all at or orders of magnitude less than tenths of µg/L. Antimony is highly variable with estimated dissolved concentrations ranging around a mg/L except where the oxidation-reduction potential becomes slightly reducing. Adsorption of antimony to either iron or manganese oxides is not possible with the current state of geochemical models. At slightly reducing conditions, the estimated antimony concentration is typically at the tenths or hundredths of a µg/L.

Table B-2 summarizes the changes in copper, lead, zinc and antimony concentrations in micrograms per liter (µg/L) as the chemical reactions progress in Lake Superior, Lake Huron and Chesapeake Bay from 0.0 to 100 percent reaction (End). As in Table B-1, both the initial total area metal concentrations and the 5 year total area metals concentrations were used in the modeling. The amount of iron oxyhydroxide was modeled in three steps; 0.3, 1.0 and 2.0 grams. Chemical reactions occur relatively rapidly with all the metals concentrations becoming detectable (0.1 µg/L) at 4 to 48 percent reaction progress in the total area.

Chesapeake Bay has the lowest estimated metals concentrations. Copper in the lakes commonly goes through a dissolution cycle that goes to, or less than, detection level concentrations by one-third to two-thirds of the reaction progress. Lead has a similar cycle trend in the lakes but not in Chesapeake Bay. Zinc has few cycles. Estimated antimony concentrations are dramatic and should be ignored because they do not reflect adsorption reactions with iron and manganese oxides. The model's estimated antimony concentrations largely reflect the solubility of antimony oxide that is unrealistic in these sediments.

Table B-1
Estimated Surface Water Concentrations
Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Area	Year	FeOOH (grams)	pH	Eh (mV)	Antimony (mg/L) [1]	Copper (mg/L) [1]	Lead (mg/L) [1]	Zinc (mg/L) [1]
Lake Superior								
Total	Initial	0.3-2.0	7.34	487	2.29E-01	1.23E-04	2.82E-03	4.57E-05
Total	5 Year	0.3-2.0	7.38	477	7.08E-01	7.65E-05	4.81E-03	2.09E-04
Lake Huron								
Total	Initial	0.3	7.31	491	2.29E-01	1.49E-04	3.12E-03	5.37E-05
Total	Initial	1.0-2.0	7.34	487	1.42E-01	1.23E-04	2.82E-03	4.57E-05
Total	5 Year	0.3	7.35	484	7.08E-01	2.08E-09	4.14E-03	2.53E-04
Total	5 Year	1.0-2.0	7.35	484	7.07E-01	1.03E-04	4.14E-03	2.53E-04
Chesapeake Bay - Cedar Point								
Total	Initial	0.3	7.99	376	1.40E-01	3.34E-07	3.46E-03	3.29E-05
Total	Initial	1.0	7.37	490	1.40E-01	2.69E-09	2.72E-03	3.72E-05
Total	Initial	2.0	8.22	336	1.40E-01	4.39E-08	3.68E-03	3.72E-05
Total	5 Year	0.3-2.0	8.22	337	6.99E-01	4.66E-03	4.04E-03	1.94E-04

[1] The surface water concentrations presented in this table were estimated using a geochemical model and were not measured in the water bodies indicated.

Table B-2

Reaction progress model results for the initial detected concentration (0.1 micrograms per kilogram), maximum concentration, drop in concentration and the ending concentration at 100 percent reaction for copper, lead, zinc and antimony.

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Area	Database	FeOOH (grams)	Antimony				Copper			
			Initial	Maximum	Drop	End	Initial	Maximum	Drop	End
Lake Superior										
Total	Initial	0.3	1.4@2%			142	0.1@14%	1.9@76%	0.1@78%	0.1
Total	Initial	1.0-2.0	1.2@2%			125	0.1@48%	0.2@71%	<0.1@77%	<0.1
Total	5 Year	0.3	7.1@1%			709	0.1@4%	12.1@67%	<0.1@71%	0.1
Total	5 Year	1.0-2.0	6.2@1%			622	0.1@19%	1.2@66-67%	<0.1@70%	<0.1
Lake Huron										
Total	Initial	0.3-2.0	1.4@1%			142	0.1@11%	2.2@76%	0.1@78%	0.1
Total	5 Year	0.3	7.1@1%			709	0.1@3%	14.3@67%	0.1@70%	0.1
Total	5 Year	1.0-2.0	7.1@1%			709	0.1@15%	1.4@67%	<0.1@70%	<0.1
Chesapeake Bay - Cedar Point										
Total	Initial	0.3	1.2@1%			125	0.1@8%	1.6@48%	0.1@50%	0.2
Total	Initial	1.0	0.1@0.1%			709	<0.1			<0.1
Total	Initial	2.0	0.1@0.6%			142	<0.1			<0.1
Total	5 Year	0.3	6.2@1%			622	0.1@2%	5.3@42%	0.1@45%	0.1
Total	5 Year	1.0-2.0	0.2@1%			709	<0.1			<0.1

All concentrations in micrograms per kilogram; Reaction progress from 0 to 100 percent.

Table B-2

Reaction progress model results for the initial detected concentration (0.1 micrograms per kilogram), maximum concentration, drop in concentration and the ending concentration at 100 percent reaction for copper, lead, zinc and antimony.

Preliminary Health Risk Assessment for Proposed U.S. Coast Guard Weapons Training Exercises

Area	Database	FeOOH (grams)	Lead				Zinc			
			Initial	Maximum	Drop	End	Initial	Maximum	Drop	End
Lake Superior										
Total	Initial	0.3	0.1@31%			2.8	<0.1			<0.1
Total	Initial	1.0-2.0	0.1@31%			2.8	<0.1			<0.1
Total	5 Year	0.3	0.1@10%	5.5@40-42%		4.8	0.1@47%	0.2@83%		0.2
Total	5 Year	1.0-2.0	0.1@10%	5.5@40-42%		4.8	0.1@47%	0.2@83%		0.2
Lake Huron										
Total	Initial	0.3-2.0	0.1@28%			3.1	0.1@98%			0.1
Total	5 Year	0.3	0.1@9%	4.7@35%	4.1@67%	4.1	0.1@41%			0.3
Total	5 Year	1.0-2.0	0.1@9%	4.7@35%	4.1@67%	4.1	0.1@41%			0.3
Chesapeake Bay - Cedar Point										
Total	Initial	0.3	0.1@25%			2.8	<0.1			<0.1
Total	Initial	1.0	0.1@10%			4.1	0.1@24%			0.2
Total	Initial	2.0	0.1@49%			3.7	<0.1			<0.1
Total	5 Year	0.3	0.1@7%			2.9	0.1@28%	0.2@82%		0.2
Total	5 Year	1.0-2.0	0.1@10%			4.1	0.1@24%	0.2@74%		0.2

All concentrations in micrograms per kilogram; Reaction progress from 0 to 100 percent.